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The Bancroft Memorial Lecture.[1]

"ORGANISM": A CONSIDERATION OF REGULATED VASCULAR TRANSPORT.

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THIS occasion has for me a peculiarly significant value. Standing before you as Bancroft Lecturer for 1937, I am able to pay personal tribute to the man in whose honour these lectures are given, in that good fortune has enabled us in Adelaide to complete one of his many investigations. Like most famous physicians of his epoch, Bancroft was a natural philosopher, and his interest and action were aroused by the use of the leaves of a shrub as a chewing narcotic by the aborigines. Bancroft classified the shrub *Duboisia pituri*, a name finally altered to *Duboisia hopwoodii* by von Mueller. He carried out extensive experiments on animals to determine its physiological action, and proved that the leaves contained a most powerful nicotine-like poison, causing death by respiratory paralysis. Bancroft was able to prove conclusively that the poison was quite different from the mydriatic contained in the leaves of *Duboisia myoporoides*. That was in 1872; in 1933 Le Messurier and I were able to establish the existence of an entirely new alkaline in the leaves of *Duboisia hopwoodii*. With the isolation and identification of dextro-nor-nicotine, we in Adelaide may justly claim to have completed Bancroft's work on the *pituri* of the Australian aboriginal, and I am grateful to your council for this unexpected opportunity to pay this personal

[1] Delivered at a meeting of the Queensland Branch of the British Medical Association on June 4, 1937.

and practical tribute to a great physician in his own city.

The title of this address called for a request for confirmation from your honorary secretary. I therefore quote the Oxford Dictionary definition of the term "organism"—"organized body, with interdependent parts sharing common life". There is no topic better suited for the illustration of this definition, and none which, in view of recent additions to our knowledge of vascular transport, better serves to show that the whole is infinitely greater than the sum of its parts—a salutary reminder in times when disease tends to be a greater subject of study than the patient exhibiting it.

It will be remembered that when Harvey deduced the existence of a circulation of the blood from simple phenomena which he demonstrated to King Charles, the existence of capillaries could only be postulated. With Leuwenhoek's discovery of the microscope, not only did capillaries swim into our ken, but the existence of highly differentiated types of cells and cell groups was disclosed. It is the relation of these cells to each other in their own struggle for existence, either singly or in groups, which makes the regulation of the transport of material to and from them a matter of great complexity and which emphasizes the meaning of the term organism.

We may classify the transport of material to and from the cells under the following headings:

On the intake side we have to consider such things as oxygen, water, salts, sugar, lipoids, protein building elements, hormones, chemical transmitters of nervous action, and the like.

On the output side, or as waste materials, we have carbon dioxide, urea, uric acid, creatinin, and other products of cell metabolism.

In addition to these the blood stream must carry, when necessary, toxins resulting from bacteriological or other action, or medicinal drugs taken by the mouth or directly injected.

A little thought will at once give rise to the question as to how this complex transport is conducted so that, for example, an excess of waste material carried in the blood does not interfere with the rejection of the same by cells less favourably situated on the blood stream. The same question arises when supply is considered: how are the demands of cells equitably dealt with on a one-way transport system which is being continuously depleted by the cells lying along its route? This is an aspect of ribbon estate development which would tax the ingenuity of any transport authority.

In addition to this material transport there is another, disclosed in recent years by the work of that master technician, Rein. The blood stream functions as a temperature equalizer, cooling those organs where oxidative processes would otherwise raise the temperature above the level of survival limits. This function, however, offers no special problems, since heat exchanges in the capillary area will be instantaneous, taking place by conduction and incurring no permeability difficulties at cellular interfaces.

We are accustomed, when considering the blood flow to organs, to focus our attention on the elementary requirements of quantity and rate of supply; these are, as you all know, determined by cardiac activity and vasomotor regulation. There still remains, however, the more important aspect of the problem—the organization and regulation of the material exchanges between blood in the capillaries and the cells of the adjoining tissues.

Let us deal briefly with the factors involved in ensuring the elementary requirements of the blood volume per unit of time in any section of the circulation. Starting at the heart, we note that the minute volume—the quantity ejected per minute from the ventricles—depends directly on the venous return. The minute volume is in fact a measure of circulatory effort. The continuous branching of the arteries, by increasing the total cross-section of the vascular bed, leads to a fall of both pressure and velocity of flow as the distance from the heart increases, until at the arteriolar-capillary junction there is a quite sudden drop in these values owing to the special throttling action of the arteriolar musculature. Thus from a velocity of half a metre per second in the aorta there is a drop to a fraction of a millimetre in the capillaries, where the slow passage of the blood gives ample time for the interchange of material across the capillary wall and cell membrane.

The changing demands, often sudden and enormous, on the part of the cells for blood supply is met by an intricate interlocking mechanism which is astonishingly perfected to meet this requirement. The minute volume can rapidly be increased by accelerating the cardiac rate, whilst the same sympathetic nervous mechanism, both hormonal and nervous, simultaneously increases the rate of flow through the coronary arteries, thus satisfying the demands of the cardiac muscle. Accelerated venous return from the systemic and pulmonary systems, partly by means of the foregoing mechanism and partly by means of increased force of contraction and output per beat, again raises the minute volume. The part played by either of these mechanisms is nicely adjusted to the capacity of the cardiac muscle, and the accelerator reflex of Bainbridge, arising from the tension of the roots of the great veins, may be viewed really as a safeguard to overfilling. The pericardium, too, the function of which was so long unanalysed, has been shown by Rössler to be a simple means of regulating the filling of the left ventricle. As both chambers occupy the same inelastic fibrous bag, the embarrassment of the left ventricle due to sudden resistance to outflow prevents by simple displacement the normal filling of the right ventricle and so regulates *in extremis* its own filling. By means of a "Zip" fastener sewn into the pericardium, Rössler was able to demonstrate this highly important and, on consideration, self-evident fact. With the pericardium open, left ventricular failure developed at raised blood pressures which had no effect with the pericardium closed. Bainbridge's reflex is not the only safe-

guard against overfilling of the right heart; and, as is shown by a consideration of the pericardial action, the blood is left on the venous side, largely in the liver, which can lodge as much as 1·5 litres, according to Wenkebach, without any disturbance of function of that organ. In man the spleen acts less in this capacity than it does in other animals. Finally, continued overloading of the heart leads to muscular hypertrophy—one of the ultimate means of coping with increased vascular demands.

We now know that there are vasomotor and cardiomotor reflexes arising from the vessels themselves, and the aortic and carotid sinus reflexes of Marey and Hering respectively are really specialized aspects of this generalized mechanism, developed in order to protect the heart itself and the central blood supply. Rise of systemic pressure in these regions sets up reflexes which simultaneously slow the heart and dilate the systemic vessels. A fall of blood pressure acts in the opposite manner. Proof that these reflexes arise also from other vessels is shown by the effect of inflating a blood pressure armlet, which, by causing collapse of the vessel walls, calls forth sufficient pressor reflex to raise the blood pressure by five to ten millimetres, whilst in cases with slight myocardial weakness the electrocardiogram will demonstrate reflex cardiomotor response. Another example of this is the fact disclosed by Rein's thermostromuhr, that within one to two seconds of ligating one carotid artery, the blood flow through the other has increased by 50% to 100%. The thyreoid blood vessels play an important part as a shunt in this connexion, being reflexly dilated by a rise of pressure in the carotids, even by the pulse waves, thus providing a protection to the cerebral circulation by means of a short circuit (Rein and co-workers). When we come to the capillaries, although there is direct evidence of nervous supply and control, we find that chemical, including hormonal and humoral, influences are most powerful. Lactic acid, for example, produced during muscular activity, dilates the arterioles in the active muscles, whilst at the same time adrenaline, which is set free from the suprarenals and at the endings of sympathetic nerves during the effort syndrome, loses its constrictor action in those very areas. It would be difficult to cite a better example of chemical coordination, which, whilst it causes limitation of blood flow in comparatively inactive areas, is prevented from constricting the vessels in active musculature. Krogh, the pioneer of the use of quantitative measurement in capillary studies, showed that the actual number of patent capillaries in active muscle increased by some twenty times, thus enlarging by two hundred and fifty times the area of capillary wall through which the carbon dioxide and oxygen interchange takes place. An excellent analogy, suggested by Krogh, is that of a ship crowding on more sail area to get speed—we take reefs out of our capillary surface for exactly the same reason, expressed in terms of energy production. Another interesting fact which should have been self-evident is that the circulating blood volume at rest is inadequate to supply oxygen at the necessary rate when the heart is doing its utmost. It was only recently, when Barcroft drew attention to the depot function of the spleen, that it became apparent that extra blood must be added to the circulating volume in exercise. In man, other depots—the liver and large veins—appear to be more important than the spleen; but all these sources of additional blood do so by diminution of their cubic content by contraction evoked by adrenaline and sympathetic action generally. The normal result is that blood is diverted from relatively inactive areas to places where it is urgently wanted, whilst the increased circulating volume provides the means for the increased rate of carriage of oxygen. In this field our knowledge is by no means complete, and it is only necessary to mention the curious effect of anterior pituitary extracts in restoring vascular adjustment to effort, such as is seen in Simmond's pituitary cachexia. Even the urinary pituitary hormone, prolan, has been shown to have this effect; whole fresh suprarenal gland itself has a similar effect in the closely related syndrome of Addison's disease and other conditions, whilst "Cortin" itself does not. Adrenaline and acetylcholine, the hormone transmitters of sympathetic and parasympathetic nervous action, have the same effects on arterioles and capillaries. Adenosine, on the other hand, which appears to be a product of muscle activity, if not of cellular activity generally, and histamine also are largely responsible for dilatation of the venous limbs of capillary loops, as well as of the venules themselves.

If we consider the veins, we find that they also are subject to reflex control of their calibre. When it is appreciated that the cross-section of the great veins at the heart is twice that of the arteries on the opposite side, it can readily be understood that a small alteration in calibre of the great veins means considerable alteration in the rate of filling of the right heart. Venomotor reflexes cooperate, therefore, to aid the venous return, and in harmony with them act the chemical and hormonal controls. All those influences involved in increasing the rate of blood supply on the arterial side—adrenaline, rise of hydrogen ion concentrations, histamine *et cetera*—cause veno-constriction, thus diminishing holding capacity and ensuring more rapid cardiac filling. This mechanism has been beautifully demonstrated by Fleisch, who showed that, even when resected from the animal with nerve supply intact, a piece of vein dilated when the blood pressure in the carotid sinus rose and *vice versa*.

The portal circulation, standing as it does between two peripheral capillary beds, namely, in the gut and in the liver, occupies a peculiarly important position relative to the venous return to the heart. In man it forms the most important reservoir for blood. According to Rein, it can withhold up to 59% of its weight of blood, and since the portal circulation provides 50% to 60% of the caval flow, its regulating effect on the latter requires no

emphasis. The blood is dammed back in the liver veins by means of a sphincter-like action of a specialized portion of the muscle coat near their exits. The existence of this function was for long denied by English workers, mainly owing to the use of different animals. In cardiac insufficiency, as I have mentioned, the liver can withhold 1·5 litres from the circulation and so relieve pulmonary congestion. The subpapillary plexus of the skin, according to Meek and Eysters, is also an important depot, and Dr. O'Connor and I have noted an important effect of this in the naked aboriginal, in whom sudden alterations in atmospheric cooling power can change the cutaneous blood volume and raise systemic pressure by as much as 30 millimetres of mercury. In the lesser circulation there is also an effective depot in the pulmonary veins. These venomotor-controlled depots are to be looked upon as factors regulating the minute volume of the heart, and they are therefore of the greatest significance for circulatory efficiency as a whole. Matheson showed long ago that the effect of a rise in carbon dioxide content of the blood stimulated the vasomotor centre directly, causing a rise of systemic pressure. If the splanchnic nerves were cut, the effect was lost, but removal of the adrenals did not influence it. If you call to mind the fact that these glands are supplied by the splanchnic nerves, you will see what a subtle selection of paths of transmission has been made by the action of carbon dioxide upon the vasomotor centre. If venous return and minute volume can be thus increased, it is valuable to compare the augmentation of these factors by injection of adrenaline itself. In this case the circulating volume is increased by reduction of the calibre of extra splanchnic vessels in the skin; for example, whilst actually in excess it withholds blood in the liver by throttling the exit from the hepatic veins.

You will agree that this necessarily brief and incomplete review of the regulation of local and general minute volume discloses a most efficient and elaborately integrated mechanism. Nevertheless there are occasions on which its perfect operation is interfered with. Psychic influences are notoriously effective, and the filling of the abdomen with much food disturbs the action of the diaphragmatic pump. Its breakdown, through abnormal demands, is well seen in traumatic shock, when over-action of the sympatho-adrenal system so diminishes the calibre of the arterioles that circulating blood volume is diminished to vanishing point.

An experiment of Rein is of fundamental importance in this connexion. He carefully removed from a dog as much blood as possible without dropping its peripheral blood pressure; then, when the surrounding atmosphere was warmed until it was the same as that of the animal itself, the blood was diverted to the skin vessels in order to lower the body temperature by radiating heat, thereby depleting the systemic circulation to the point at which the venous return was inadequate. The result was syncope due to loss of circulating blood volume.

So, despite the vital necessity for the maintenance of a good systemic pressure, essential alike for coronary and cerebral circulation, the paramount reflex to maintain optimum body temperature bleeds the animal into its own cutaneous vessels. There is a possible phylogenetic significance in this phenomenon, as suggested by Rein. Vessels which subserve heat regulation by vaso-dilatation, cannot be constricted in order to raise blood pressure, possibly because most vital physiological chemical processes have an extremely narrow temperature range. Collapse in a warm bath after a heavy meal is probably to be explained on these grounds. Another interference with regulation is shown when, during muscular activity, carbon dioxide is inspired. The muscular activity produces lactic acid and localized vaso-dilatation; the carbon dioxide, acting centrally, as previously mentioned, causes vaso-constriction. The latter action is prepotent and the muscles are depleted of their blood supply. On the other hand, the local blood supply to muscles cannot be interfered with by reflexes from the carotid sinus, which, be it noted, works through control of the suprarenals as well as by vasomotor action.

The greatest wonder of the circulatory mechanism is not, however, this amazing regulation of supply of blood in adequate quantity wherever needed; it is the adjustment of material interchange between blood and tissues which, when examined, causes us to marvel and to grasp the significance of the title word of this discourse.

Consider the transport problem raised by the formation of an appendicular abscess. If we reason without knowledge of a circulation of the blood, we will be forced to conclude that any poisons produced by the infection will, if excreted in the urine, reach the kidney by some direct path of obscure, if effective, nature. This in effect was the direct result of Galen's influence, the tissues being conceived as a labyrinth without directional structure. Harvey's discovery reaches farther than we commonly realize, and, among other things, makes such reasoning impossible. But let us not be too glib in our deductions of what does happen to the supposed toxins. They reach the kidney, it is true; but how? Taken up by the mesenteric veins, you answer, they pass to the portal circulation, to the cava and right ventricle, thence through the lungs to the left ventricle, and finally by the renal artery to the kidney. This in fact is just where a certain blindness in us all becomes evident—the path described is simple, but the transport of the toxin is far from simple. It reaches the kidney finally by blind chance, for the kidney, despite the fact that the whole circulating blood volume passes through it in twenty-five minutes, lies on a side track, and it does not by any means follow that this volume of blood is in fact the whole actual circulating blood. Any given corpuscle, to take a unit of it, may circulate for days before getting into the renal side-track. The same reasoning applies to any given portion of the toxin. Another important feature overlooked in the first glib answer is that since the kidney lies

on a side-track, and since the toxin in this hypothetical instance must pass through the liver, it has an excellent opportunity to damage all the cells of the body, the liver in particular, before chance finally favours its removal. Here we are face to face with an intrinsic disadvantage of a one-way circulatory system, which no study of its gross dynamics would disclose. Products of either metabolism or pathological change in any one situation reach their final goal only after much loss of time and waste of circulatory effort. The liver, in so far as its functions are excretory, lies with respect to the main circulatory path, like the kidney, in a side-branch, and the necessity for organized transport of rejected material, bilirubin for example, from the systemic capillaries, is further emphasized by the fact that it can reach the gall bladder only after passing via the capillaries in the gut wall. Here is a second intimate contact with tissue cells, and unless the bilirubin is in some manner bound in the blood, it could pass outwards and interfere with cellular function in this situation.

To illustrate the importance of being on the main circulatory track we might consider the effect of a patent *ductus arteriosus* or of an Eck's fistula. The former places the lungs in a side-track, causing the insufficient oxygenation which is the main pathological result; the latter places the liver in a side-track with respect to the portal blood and so far reduces the chance of bile pigment reaching the gall-bladder that urobilinogenuria results. It is well known that poisons absorbed from the gut are likely to damage the liver—after an Eck's fistula is established, ingested phosphorus, for example, does not affect the liver.

With the exception of the lungs and the liver (only so far as the blood returning from the gut is concerned) there is then no directed blood flow in the sense that it is forced to go direct to the organ with which it has to deal. If a particular tissue or organ rquires a certain foodstuff—say glucose—the whole blood must have its sugar concentration raised. This means that five litres of blood must have its glucose concentration increased. A direct supply of glucose to the organ in question is impossible, and all other tissues are necessarily subjected to the effect of that increase. The same is true with regard to waste products of metabolism, which must continue to circulate in rising concentrations until they are finally removed, giving them ample opportunity to attach themselves to tissues for which they may have affinity.

Consider, too, the transport in each capillary loop. Cells on the arterial limb have an advantage on this one-way system over their neighbours farther down stream, both with regard to intake of oxygen and other necessaries, and with regard to excretion of waste. It is, however, extremely unlikely that a multicellular system could develop except along lines of organization so as to obviate these difficulties. And here we find the meaning of the term "organism" illustrated in the most fundamental manner, that is, in terms of physical chemistry of molecular aggregates and surfaces. The wonderful adaptation of the hæmodynamics to the gross problem of volume supply would be of little value but for this ultimate molecular aspect of living organism.

The carriage and exchange of oxygen and carbon dioxide serve as a good example of organized and regulated transport. The lungs lie on the main stream; the flow from thence is direct to the tissues. Only 1·3% to 1·5% of the oxygen is actually carried in the dissolved condition, the remainder being combined with hæmoglobin in the corpuscles. There is, however, a chemical equilibrium between the small dissolved fraction and the combined oxygen. As the tissues take up oxygen from the dissolved fraction, it is automatically replaced by oxygen from the corpuscles, so that the dissolved fraction is always at the same concentration, or, which is another way of saying the same thing, at a constant pressure. In this manner the pressure head of oxygen is maintained throughout the length of the capillary loop and all the bordering cells get an equal chance of securing their supply. The corpuscle, it will be noted, is a vehicle for transporting oxygen, so constructed that a constant pressure of dissolved oxygen is maintained in the plasma water. The mechanism of this equilibrium is highly complex. Dissolved hæmoglobin would be quite valueless for the purpose. The concentrated mass of hæmoglobin which it is possible to pack into each cubic millimetre of blood depends on the corpuscular method of carriage. The lipoid envelope is such that the potassium which was bequeathed from the normoblast cannot diffuse out, whilst the chlorine of the sodium chloride in the plasma can readily diffuse in. Since the hæmoglobin is imprisoned within the corpuscle, changes in available ionized chlorine in the plasma cause it to form reversible combination with hydrogen, so setting free sodium ions for combination with carbon dioxide. In other words, the parcelling up of the respiratory pigment in a lipoid semipermeable membrane at once confers the properties requisite for an organism, for by its means the rate of liberation of oxygen and absorption of carbon dioxide by the blood (and *vice versa* in the lungs) are adjusted nicely to local requirements. The shape of the corpuscles is such that it provides the greatest possible surface for ionic and gaseous interchange, the total surface having been calculated to be 3,200 square metres. To pass the capillaries they are of the same order of diameter, but when the latter become constricted ever so slightly, the resistance to onward flow rises rapidly, since the corpuscles then become deformed in order to pass. In this manner a control of flow in the capillaries is secured by means of these endothelial tubes in a manner which, until 1920, appeared to be an anatomical impossibility.

There are sufficient of these oxygen vehicles to enable the venous blood to return with a load of oxygen varying from 70% of its original amount, at rest, to 30% in very severe exercise. Among the properties of hæmoglobin which may appear to be

of more academic interest is its more rapid decomposition with rise of temperature. This is very important, for where tissues are actively utilizing oxygen the temperature rises rapidly, providing another example of organization for a given end.

Consideration of a case of congenital cardiac disease will provide illustrations of the cardinal features of the transport regulation as it affects oxygen and carbon dioxide. The shunting of the lungs from the main into a side-track of the circulatory pathway leads to the supply of mixed arterialized and venous blood to the periphery. The low oxygen tensions thereby produced in the sinuses of the bone marrow lead to accelerated hæmopoiesis, and the resulting polycythæmia increases the viscosity of the blood in cases we have had under investigation by as much as four times. There results therefrom a much slower flow in the capillary loops, which favours the unloading of oxygen at the lower tensions resulting from arterio-venous mixing. At the same time the extra buffering due to the carbamino compound formation by excess of hæmoglobin provided by the polycythæmia quite counteracts the effects of loss of base due to the hyperpnœa present in such cases.

Although carbon dioxide is thirty times more soluble in the water of the plasma than oxygen, and diffuses twenty-five times as rapidly, its regulated carriage for physiological purposes almost entirely depends upon the parcels of hæmoglobin in the blood, mainly owing to the equilibrium which produces free base in the blood, as previously mentioned, but also to the direct production of a carbamino compound between carbon dioxide and the amino groups of the hæmoglobin complex. Calculation has shown that even with all this provision for regulated gaseous transport, the speed with which carbon dioxide is taken into or yielded from solution in the plasma would be too slow for physiological purposes. Roughton has recently shown by measurements of reaction velocities that this is achieved by the action of an enzyme—a dehydrase—which accelerates the process to meet requirements.

Even the water of the plasma is carried in packets analogous to the corpuscles. The major portion of the water is transported in the form of water of imbibition of the colloidal particles of serum protein. It is in equilibrium with the smaller fraction of free water, and the quantity of the latter varies, among other things, with the blood pressure. Rising pressures set free more water; falling pressures lead to more imbibition by the protein. If the blood pressure falls extensively, as after hæmorrhage, the imbibition pressure in the capillaries may exceed the blood pressure, and water is drawn in from the tissues to make up circulating volume. A simple fluid circulation would not serve the varying biological needs of the body—a point which may well be borne in mind in connexion with present-day tendencies to give excessive volumes of continuous intravenous injections of saline solution. Without the necessary serum albumin vehicle for transport of the water, it will leave the vessels in company with diffusible salts, emptying the vascular system of circulating fluid, thus producing general tissue œdema. This latter in fact does largely result in many cases of continuous intravenous administration of saline solution, where there is more water injected than there is protein to carry it. It is almost too easy to make an intravenous injection, and our conceptions of what happens when we do are as vague and numerous as are conceptions of heaven. The gaseous and water transport just described is general in its regulating mechanism, while, on the other hand, the transport of most other materials is more complex, both in the carriage and the unloading thereof. Take, for instance, the carriage of a dye foreign to the organism, when injected into the blood stream. Either the kidney or the liver provides the exit for such substances in the urine or bile respectively. The problem for the organism is to transport the substance in some form which prevents its action on the tissues and yet in a form from which it can readily be released in the appropriate excretory organ. Congo red provides a good test substance. It has a great avidity for tissues, which it stains; yet when injected intravenously it is locked up in the blood until it is finally excreted. So far as the circulatory system as such goes, it can continue to circulate the substance only until it can ultimately reach the excretory organs, but it is in the vehicular method of carriage that the secret of regulated transport of such substances lies. It may be thought by some that the secret would lie in the impermeability of the cell membranes to foreign substances, but we know from experiments on the vital staining of protozoa that this is not so. The injected dye is immediately bound to the serum protein, which so protects it that it circulates through the capillary bed without passing outwards to the tissues. Not being in aqueous solution, but in the form of a compound of dye and protein, its physical and chemical identity being lost, according to Bennhold the dyes become helplessly attached to the protein. So far, so good. The protein vehicle has protected the tissues adequately, but what of the excretory phase? How is the dye ever set free? When the protein-dye complex reaches the liver, the dye is set free and excreted in the bile. So, during the roundabout circulation imposed by the one-way stream, the dye is bound until, bit by bit, as the loaded protein vehicles, at last in their turn, reach the portal circulation, the foreign substance is unloaded from the vehicles and removed from the blood. The process is analogous to that by which hæmoglobin picks up oxygen in the lungs and yields it only to the tissues which require it. In discussing this I drew attention to the enormous corpuscular surface area of 3,200 square metres which is available for gaseous exchange. The surface which is available for binding foreign substances, such as the Congo red in this instance, is no less than 140,000 square metres.

We are indebted to Stanford Rosenthal, who was one of the first to demonstrate this protein-dye combination and who also indicated the nature of the unloading process. In the liver, as he pointed out, the bile acids are produced, and these lower the surface tension at the interfaces of the colloid particles of the serum protein, thus setting free the adsorbed dye. Bennhold, who has confirmed this, has experimentally shown that the same method is used in the transport of bile pigment. In the kidney a similar mechanism has been shown to operate, eosin being unloaded and excreted in the urine.

Bilirubin, as you know, is formed not in the liver, but in reticulo-endothelial cell tissue throughout the body. Let us trace the excretory path of bilirubin formed in the marrow of a rib. We know that its concentration in the blood is low and that its affinity for tissue cells is great. The circulation of a given bilirubin moiety will carry it many times round the whole system before it ultimately passes into the mesenteric arterial system, the capillaries of the gut, and thence into the portal vein and liver capillaries. Clearly the chance of the bilirubin's leaving the capillaries in the gut to combine with surrounding cells must be very considerable. Being bound, as it is now known, to serum albumin, it cannot be unloaded until it reaches the liver itself by means of the surface tension phenomenon already mentioned. The extent to which the liver is placed in a circulatory side-track is clearly demonstrated by the work of Rein and his colleagues, who proved that only 13% to 25% of the circulation of that organ comes from the hepatic artery. The bilirubin-albumin complex, however, will circulate indefinitely without setting the bile pigment free until it comes to the liver. The globulins, which play no part in this transport, are involved in the conveyance of lipoids and lipoid-soluble materials, such as carotin.

Electrophoresis of dye-coloured serum protein leads to the separation of an albumin-dye compound which moves to the positive pole more rapidly than does the globulin. Analysis then shows that cholesterol is bound to the globulin. If a fat-soluble dye, such as Sudan III, is added, it is bound to the globulin in combination with the cholesterol. The function of globulin as a vehicle is highly significant in diseased conditions and in certain states in which the kidney was formerly considered to be the blameworthy organ. Hicks and Mitchell have demonstrated that in a case of nephrosis in which the urine "went solid" on being allowed to stand, the phenomenon was due to the separation of a fine reticulum consisting of a globular lecithin complex. The loss of carbon dioxide caused the pH variation necessary to displace the complex from solution in a clear straw-coloured urine. Clearly this was a case in which the capsule of Bowman passed a soluble globulin-lipoid complex as a gross indication of disturbance of the transport system, possibly as a secondary manifestation of some metabolic upset.

The enormous surface available for adsorption may make unnecessary any other explanation of the mode of carriage of dyes and other substances. The question of an equilibrium such as exists between corpuscular oxygen and dissolved plasma oxygen probably does not arise. Certain it is, however, that in respect of combining power for dyes the serum proteins vary in different pathological subjects. Bennhold has repeatedly shown this for naphthol yellow. In this connexion our case of nephrosis is of especial interest as an instance of disturbance of the transport (vehicular) function, because it has been proved that in nephrosis of this type the blood is unable to bind firmly any added fat-soluble dye. It is just in such cases as those of nephrosis that when there is a cholesterinæmia there is also a globulinæmia. This suggests that when there is a pathological demand for more of the vehicle for transport—in this case of lipoid—it is forthcoming. We find this to be the case in pregnancy, nephrosis and xanthoma; whilst in chronic jaundice it is the albumin, that is, the bilirubin carrier, which is increased. Uric acid is carried partly bound to albumin and partly in solution, an equilibrium existing between the two fractions. Salicylic acid in therapeutic amounts is similarly carried by the albumin, as also are "Neosalvarsan" and "Atebrin". If such drugs are injected too rapidly or if the serum composition is altered, unbound drug circulates in the blood stream with in extreme cases quite different pharmacological results. With substances like lithium carmine or "Thorotrast" the unloading factor is found to reside in the reticulo-endothelial system, which removes them from their protein carriers and adsorbs them in its own cellular structure.

The proteins of the blood then, far from being present for some vague nutritional purpose or for contributing to the viscosity of the blood, as the text-books often state, have a function of immense importance to the organism as a whole. They illustrate in fact that very definition with which I began this involved discourse. By their nature they at once order the arrangement of substances for carriage in the blood stream into free, part-free and part-bound and entirely bound.

I hope that this very imperfect presentation of the subject will have made clear at one and the same time the implications of the one-way circulatory system and the vital necessity for organization of transport in the blood stream itself. If in addition this analysis of the circulatory and transport mechanisms within the body serves to press home important clinical implications—among which none is more important than the conception of organism—it will have achieved not only its physiological purpose, but a philosophical one very germane to an address to medical practitioners by a medical experimenter, namely, that there exists really no essential conflict between an explanation in terms of causality and one in terms of finality.

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PEPTIC ULCER.[1]

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IN preparing this evening's paper I have sought to base my remarks on an analysis of the patients suffering from peptic ulcer admitted to the Adelaide Hospital during the last six years or so. This has meant surveying over one thousand case folios, of which many have been rejected on account of containing insufficient or ambiguous data. Those retained number 644 folios, and their contents have been judged by myself to be trustworthy and a reliable source of information for the purposes of this paper.

The subject of peptic ulcer is such a vast one that in the time available it will be possible to deal only with some special aspect, and I have thought that of treatment to be the most suitable.

Peptic ulcer occurs in those parts of the alimentary tract bathed by the gastric juice, and anatomically may be divided into œsophageal, gastric, duodenal and anastomotic. The first named is so rare that it need not be considered, while the last occurs in the region of the stoma of such an operation as gastro-enterostomy. The above varieties may be further divided into acute, subacute and chronic. This division is based on not only the duration of the symptoms, but also upon the histological and radiological appearances of the ulcer. For this purpose the duration of symptoms is a somewhat unreliable guide, for some ulcers, chronic histologically, may give no trouble until they perforate acutely. Furthermore, the fact that an ulcer is discovered by means of X ray examination does not necessarily mean that the digestive symptoms complained of are due to this ulcer. It is one of the misfortunes of modern medicine that an X ray shadow is often regarded as being of more value in diagnosis than the substance of sound clinical history-taking and observation. I have treated the scar of an old ulcer, only to find later that the symptoms were due to some other pathological condition, and that may be an experience that others have had.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 29, 1937.

Pathology of Peptic Ulcer.

As the treatment of peptic ulcer depends upon the pathological changes present, a short account of these is necessary.

The acute ulcer, whether arising from the breaking down of a primarily necrotic area or resulting from some local vascular disorder, rarely penetrates further than the submucosa. In the early stages, with the naked eye, an area of mucosal necrosis with raised edges is seen, stained black by altered blood in the stomach; in the duodenum the area is similarly black or on occasion bile-stained. Later this slough separates, leaving a small depression with clean floor and sides that may overhang. In the earlier stages hæmorrhage from the exposed vessels may occur and is the cause of many cases of melena and hæmatemesis for which no radiological change can be found. As a rule, the acute ulcer heals quite rapidly and ultimately no evidence of its presence may be found. On the other hand, the larger acute ulcer may extend to involve the deeper layers of the organ, to become a subacute or a chronic ulcer, or even to perforate. It is quite reasonable to suppose that the common chronic ulcer began as an acute ulcer which failed to heal.

These acute ulcers are usually multiple, and in the stomach may be present by the hundred; some authorities prefer to call them "multiple erosions". There is a definite association between the onset of acute ulcer and the presence of areas of focal sepsis. Of these, the most frequently found are upper respiratory infection, appendicitis, cholecystitis and dental sepsis. As a terminal event "multiple erosions" may be met with in peritonitis, uræmia and malignant disease.

The subacute ulcer constitutes a stage intermediate between acute and chronic ulcer, and is very difficult to recognize clinically. It occurs most often in the stomach and may be single or multiple. To the naked eye this variety is often quite deep—not so much from actual destruction of the wall of the organ affected as from a heaping up of the edges, the result of inflammatory changes. The ulcer normally extends to the muscular layer which forms its floor. Histologically there is considerable fibroblastic response; it thus differs from the acute type, in which more active inflammatory changes are found. The subacute ulcer may heal, to leave an easily recognizable scar, or may progress to form a chronic ulcer.

The chronic ulcer is almost always single in the stomach, although the scars of healed ulcers may be found near a more active lesion. In the duodenum "kiss" ulcers may be found, that is, one on the posterior wall with another on the opposite part of the anterior wall. Nevertheless, both in stomach and duodenum, chronic ulcers are mainly solitary. Simultaneous occurrence of these ulcers in duodenum and stomach may be seen.

Regarding the site of chronic gastric ulcer, all authorities agree that the majority occur along the lesser curvature, more being found at the pyloric end than elsewhere. Even when they are

on the posterior wall, the tendency is for them to be near the lesser curvature.

In the duodenum chronic ulcer is almost confined to the first part, and there almost entirely to the anterior or the posterior wall or both as "kiss" ulcers. Their distance from the pylorus is variable, and sometimes they encroach upon the pyloric canal. The sizes of chronic ulcers vary from large ones, over an inch in area, to small ones, not as large as a split pea. Gastric ulcers as a rule are much larger than those of the duodenum. In the duodenum the largest ulcers are found on the posterior wall.

The shape and depth vary with the situation and age of the ulcer, and also with its progress or healing. Some have overhanging walls, so that the mouth of the ulcer is much smaller than its floor, while others have sloping walls. The surface surrounding the ulcer becomes œdematous and angry-looking from inflammatory reaction: a true gastritis or duodenitis.

All chronic ulcers invade the muscular layer, many invade the serous coat, and some penetrate surrounding organs, such as the pancreas and liver. Their walls become tough from the fibrous and granulation tissue formed by the processes of repair. The area around the serosal aspect of chronic ulcer is often surrounded by adhesions, the result of local reaction on the part of the peritoneum; these bind the affected area and its surroundings to neighbouring parts.

Changes in the blood vessels of the ulcer region are important. As has been indicated above, in acute ulcer bleeding from damaged vessels is common and is a fertile source of melena and hæmatemesis. In chronic ulcer the process is not so rapid, so that blood vessels have time to become closed by thrombosis or obliterating endarteritis. For these reasons bleeding in chronic ulcer is not so frequent as might be expected. Nevertheless, severe and sometimes fatal hæmorrhages do occur from the erosion of an artery by the ulcerative process. Healing occurs in chronic ulcer with the formation of a depressed scar, often having radiating bands leading towards the centre, thus giving a puckered appearance to the cicatrix. This puckering is almost always confined to gastric ulcer and may lead to the formation of hour-glass stomach when the original ulcer has been large. The scar of chronic ulcer is easily detected from the serosal aspect, thus differing from those of acute and subacute ulcer, which are not so recognizable. It would appear that chronic ulcer of the stomach heals more readily than duodenal ulcer, although this still remains a matter for argument. Nevertheless, the fact remains that chronic ulcer of the duodenum may heal, and without the aid of a gastro-enterostomy operation. Healed duodenal ulcers are found at autopsy, and not uncommonly.

Apart from the changes mentioned above, there are certain conditions arising from chronic ulcer that are of the greatest importance. Hour-glass contraction of the stomach has been mentioned already, and curiously enough is almost entirely confined to females. Stenosis of the pyloric canal and of the duodenum from scarring is a frequent and unfortunate result. In this respect it should be remembered that the residual narrowing after suitable treatment has been carried out is often not nearly so great as that indicated by X rays or by clinical observation in the untreated case. The initial narrowing is due to a combination of three factors: cicatricial stenosis, inflammatory œdema and muscular disorder. The last two can often be reduced by treatment; the important bearing of this fact on treatment will be indicated later.

Chronic ulcer is always accompanied by a widespread gastritis or duodenitis, or both, which, apart from confusing the clinical history and picture, makes the ulcer appear to be bigger than it really is when investigated by the radiologist. It also temporarily narrows the lumen of the pylorus and duodenum. This gastritis and duodenitis are due partly to an invasion of the superficial layers by inflammatory cells and partly to a lymphoid hypertrophy. These changes may impede the normal secretion of the organs affected.

Peritoneal adhesions resulting from chronic ulcer have already been discussed.

Reference to Table I will indicate the anatomical distribution in the two sexes in the series studied. It will be seen that duodenal ulcer is more common than gastric ulcer in both sexes, but that gastric ulcer in the female is slightly more usual than is duodenal ulcer. This distribution does not vary appreciably from the figures of other published series.

TABLE I.

Distribution of 550 Cases of Peptic Ulcer.

Site.	Male.	Female.	Total.
Gastric:			
Just stomach	1	2	3
Lesser curvature, high	14	4	18
Lesser curvature, middle	13	2	15
Lesser curvature, low	38	4	42
Just lesser curvature	13	4	17
Prepyloric	48	11	59
Greater curvature	5	3	8
Total gastric ulcer	132	30	162
Duodenal	299	58	357
Pyloric (all at operation)	10	—	10
Anastomotic:			
For duodenal ulcer	9	2	11
For gastric ulcer	1	2	3
? Cause	3	1	4
Duodenal and gastric	3	—	3

Effects of Ulcer on Motor and Secretory Functions.

A consideration of the influence of chronic ulcer on motor and secretory functions is most essential so far as arriving at a sound basis for treatment is concerned.

Probably the most important is the alteration of the activities of the pyloric sphincter. This sphincter may be affected in two ways. First, a reflex lack of opening or relaxation of the sphincter may occur when the highly acid gastric contents pass on to the region of the ulcer in the duodenum. This event usually happens when digestion has been in progress for an hour or so. Secondly, when the ulcer, either duodenal or gastric, encroaches upon the pyloric area, a true pyloric spasm may be set up. In either case, a slowing up of the onward passage of food through the pylorus occurs and in time may lead to the occurrence of gastric atony and dilatation. Of course, diseases other than ulcer may bring about a similar state of affairs in the pylorus; for instance, gall-bladder, appendicular or intestinal disorders, such as diverticulosis. Furthermore, the hold up in the evacuation of stomach contents through the pylorus results in an increasingly greater proportion of acid to food in the stomach. This is brought about by two factors: first, the food remaining stimulates the stomach to go on secreting more acid, and secondly, the usual reflux of alkaline duodenal contents is delayed. This explains the "climbing" test meal curve so often obtained in such cases.

Medical Treatment of Peptic Ulcer.

It may safely be said that there is no treatment that is applicable to all ulcers. So far as treatment is concerned, each ulcer is a law unto itself. Further, once an ulcer, always an ulcer; for, even if the ulcer does heal, the underlying diathesis remains and has to be treated. Otherwise there is every likelihood of recurrence.

Nevertheless, although the details of treatment vary, the underlying principles remain the same. Briefly they may be stated to be: (i) physical and mental rest; (ii) a diet chemically and mechanically non-irritating; (iii) clearing up of septic foci; (iv) alkalinization of the gastric contents and the use of drugs to diminish secretion and relieve "spasm"; (v) continuation of this treatment until such time as the indications are that the ulcer has healed; (vi) suitable after-treatment.

Dealing with the principles in the order mentioned above, rest is most important. This requires a period of constant stay in bed in congenial surroundings. It is desirable that the patient be kept warm and in a more or less constant temperature. It is well known that changes in temperature may aggravate symptoms or even initiate them. The physical rest should be absolute—I have known of patients being allowed out of bed to go to the lavatory or the bathroom, or even to take a drive in a motor-car. As far as is possible, ulcer patients should be freed from business or other worries and prevented from becoming bored with their lot. Most ulcer patients are active individuals, with the "live-wire" temperament, who find it difficult to live a vegetating existence.

The diet should be such that it will not irritate the ulcer surface, as would such articles as bits of crust, pips from fruit, badly cooked vegetables, tough meat and so on. Further, it should contain no articles that would stimulate excessive gastric secretion, such as condiments, underdone meats and the like. Undoubtedly milk should form the backbone of such a diet; but, as milk is really a solid food, for as soon as it reaches the stomach quite large curds are formed, it should be citrated. The citrate of soda should be used in the strength of two grains to each ounce of milk; this will prevent the formation of large curds. To increase the caloric value of the diet, cream and sugar may be added to the milk. To prevent monotony, the milk may be flavoured with tea. The twenty-four hour intake of milk should be about two to three pints of whole milk. In addition to the above, certain cereals taken with milk are allowed, such as arrowroot, maizena, cornflour, semolina or Benger's food. After a while, such vegetables as mashed potatoes and vegetable marrow may be allowed. Fruit or milk jellies with cream form a useful adjunct to the above diet.

Various *régimes* or diets have been devised to meet the above requirements, and since their originators published them they have been modified to such an extent that in all probability their authors would not recognize them as being their productions. In most cases the alteration has been in the direction of making them more liberal and this has not helped the healing of the ulcer.

Of more recent years the most popular diets have been those of Hurst, Sippy and MacLean, and in this respect it is extraordinary how few text-books, either medical or nursing, give a detailed account of these *régimes*.

In those of Sippy and MacLean the diet is built up on a "ladder" scheme so that it becomes more liberal as time goes on. Hurst strongly criticizes this idea, stating that the ulcer needs as much protection in the fourth week as it does in the first, even if it is healing; and surely one must agree with him. All of them aim at leaving the patient with something in his stomach during the whole of the waking hours and during as much of the sleeping period as possible.

This diet combines with and renders harmless the free acid so necessary for the production of an ulcer. Further, it is a diet that does not stimulate the secretion of excess of acid.

Another physiological fact that is made use of in this treatment is the power of fatty foods to decrease the gastric secretion. With this end in view, cream and olive oil are used, and furthermore they increase the caloric value of the diet.

Summaries of the *régimes* of Hurst, Sippy and MacLean are given for purposes of comparison.

Hurst's *régime* is as follows:

1. Every alternate hour from 8 a.m. to 10 p.m., five ounces of milk. This can be warm or cold and may be flavoured with tea.

2. Every other hour, alternating with 1, from 9 a.m. to 9 p.m., a five-ounce feed, which may be made of any of the following: (a) Arrowroot, cream of wheat, Benger, junket, custard. To any of these red currant, apple or

other fruit jelly can be added, and the junket may be flavoured with chocolate. (*b*) At least two should consist of thick soup or semi-solid *purée* of potato, artichoke, cauliflower or parsnip. A little sugar may be added to the (*a*) feeds and a little salt to the (*b*) feeds if desired.

3. A rusk with butter should be eaten with three feeds. Small quantities of water or sweetened orange juice or water may be drunk between feeds.

4. One ounce of cream should be added to the 11 a.m., 1 p.m. and 5 p.m. feeds, and half an ounce of olive oil should be taken before the 9 a.m., 2 p.m. and 7 p.m. feeds.

5. One drachm of Mixture 1 should be added to each milk feed, and one or more additional drachms of *Emulsio Magnesiæ*, according to the state of the bowels, are taken immediately before each feed.

6. One drachm of Mixture 2 before the 8 a.m. and 3 p.m. feeds and two drachms before the 10 p.m. feed.

7. One drachm of tribasic magnesium or tribasic calcium phosphate with a little water after the 3, 7 and 10 p.m. feeds. An extra powder should be taken after any other feed during the day or night if the slightest indigestion or heartburn occurs.

8. Wash the mouth out after each feed and thoroughly clean the tongue by scraping with a spatula morning and evening.

9. Smoking is not allowed during the strict treatment.

10. During the night the patient should have a feed with Mixture 1 by his bedside, so that whenever he wakes, whether in pain or not, he can take a feed. This can be repeated at intervals of an hour or more whenever he is awake during the night.

Mixture 1.		*Mixture* 2.	
Sodii Citratis ..	15 grains	*Atropini Sulphatis*,	$^1/_{100}$ grain
Aquam ad	1 drachm	*Aquam ad*	1 drachm

The dose of Mixture 2 should be increased if no dryness of the mouth or difficulty in visual accommodation occurs.

Sippy's *régime* is as follows:

1. Rest in bed for three or four weeks; no active work for the following three or four weeks at least.

2. Alkalis consisting of combinations of calcined magnesia and sodium bicarbonate are given half an hour before the first feeding and every hour thereafter until the last feeding, and sometimes until 10 p.m., alternating with bismuth and bicarbonate powder.

3. At 10 p.m. the stomach is relieved of all acid and other contents by means of a stomach tube. The process may be repeated during the night.

4. Milk and cream (in equal parts) are given hourly in three-ounce portions from 7 a.m. till 7 p.m.; this is continued for two or three days.

5. To the above milk and cream *régime* are added gradually soft cooked eggs and well-cooked cereals, until at the end of ten days the patient is receiving daily not only the milk and cream mixture as above, but the soft cooked eggs (one at each of three meals) and nine ounces of cooked cereals (three ounces at each of three meals), the cereal and egg meals alternating.

6. Cream soups, vegetable *purées*, custards and other soft foods may be added or substituted, never allowing more than a total of six ounces in volume at any one feeding.

7. In the fourth week the milk and cream mixture may be increased to five ounces and a two-hour feeding schedule begun. After a few more weeks the three-hour schedule will suffice.

8. With some few additions, such as bread, butter and meat, and the decreasing of some of the alkali, this diet is continued for a year or more.

MacLean's *régime* is as follows:

First week: The patient is kept on a fluid diet consisting of milk or milk and Benger's food. About three pints of milk are taken. Eight ounces or so of milk are taken every two hours. To prevent curd formation in the stomach, ten grains of sodium citrate are added to each glass of milk. One teaspoon of one of the alkaline powders described is taken every two hours shortly after the milk; about six or seven such powders are given during the day. In addition to this, a double dose is given last thing at night, immediately before retiring. The patient is warned to take some extra powder if he wakes up at night, especially if any discomfort is present. Adjustment of the powder or addition of magnesia cream may have to be made if the bowels are disturbed to an excessive degree.

Second week: If the patient is complaining of any discomfort and the symptoms still persist at intervals, the procedure for the first week is kept up for a few days longer. If there are no symptoms, the powder is reduced to four or five doses a day, the quantity of milk being somewhat reduced also. Two or three eggs are now added to the milk and beaten up. After this a small amount of toast and butter is taken and the diet gradually increased, so that by the end of the week the patient is taking, in addition to his milk, a certain amount of toast, butter, eggs, custards and cream. A dose of powder immediately before retiring is taken as before, and the same care during the night is still necessary.

Third week: The powder is reduced to three or four doses per day and one at bed-time. Food is now gradually increased and should consist largely of eggs, cream, toast and butter, custards, cereal puddings, a little white fish and a small amount of potatoes.

Fourth to sixth week: Milk is given up or considerably reduced and powders are reduced to three each day, with an extra dose at bed-time. Food is given as before, with a little pounded chicken added. In the fifth week a little meat may be taken, but only a very small amount. By this time the patient is taking a simple, nourishing diet, and the ulcer should be healed or partly healed.

Septic foci, such as dental infection, sinus infection, tonsillar sepsis and so on, should be dealt with as soon as it is convenient. The first named should be dealt with at once, but care should be taken that a suitable denture be supplied if wholesale extractions are done. Such conditions as chronic appendicitis and chronic cholecystitis need attention at a later date.

Nearly all duodenal ulcers and many gastric ulcers are accompanied by an excessive amount of acid in the stomach. It is one of the principles that this excess should be neutralized by alkalis. Many alkalis or combinations of alkali have been used and their effectiveness as neutralizing agents vary within large limits. Furthermore, some are more rapid in their action than others. The experiments of Hurst and his co-workers have shown the relative values of the more commonly used agents. From this it will be seen that the more soluble magnesium salts are the most effective, both as regards rapidity of action and neutralizing power. Bicarbonate of soda lies in the middle zone, while the bismuth salts and the aluminium proprietary and expensive products "Neutralon" and "Alocol" run a pretty poor last. It would seem that the ideal would be a combination of a rapidly acting, efficient salt, such as carbonate of magnesia, with a more slowly acting and efficient one, such as the tribasic phosphate of lime or magnesia; actually, this is the combination recommended by Hurst. One can see little advantage in using two separate powders, such as the "Pulv. A" and "Pulv. B" of the Adelaide Hospital Pharmacopœia, the former containing ten grains each of bicarbonate of soda and magnesia, and the latter ten grains each of sodium bicarbonate and bismuth carbonate. Nevertheless it is extraordinary

how patients still fly to the baking soda tin in the kitchen for relief—perhaps its rapid action and its production of something objective in the way of "wind" may explain this fact. Of course, it may be argued that one should not aim at complete neutralization, for this would disarrange gastric digestion, but in practice this is hardly likely to happen.

Belladonna and atropine are drugs that, owing to their action in reducing gastric secretion and in reducing "spasm", are of value in the treatment and are extensively used. Especially are they useful at the end of the day, to diminish the night secretion of gastric juice and so minimize the risk of night pain and irritation of the ulcer. The atropine is used in doses of one two-hundredth to one one-hundredth of a grain, given by mouth, or tincture of belladonna in doses of five to ten minims. These are given three times a day, with a double dose at night. Of course, care should be taken that visual accommodation is not disordered.

The minimum length of such treatment should be four weeks; longer if it is judged that healing has not progressed sufficiently. In an uncomplicated ulcer relief from symptoms should ensue within three days of the start of treatment. In a way this is unfortunate, for some patients are inclined to think that they are cured once their symptoms have cleared up. If symptoms do not clear up in this time, some complication should be suspected or perhaps some inefficiency in treatment, or even a wrong diagnosis. Whether the ulcer has healed or not may be judged, first, by the test for occult blood; if carried out under suitable conditions, and if the result is positive, then the ulcer is still oozing blood and so not healed. Secondly, an X ray examination should be made during the fifth week of treatment. The absence of the signs of active ulcer, such as irregular peristalsis, spasm of the pyloric antrum and tenderness over the ulcer region suggest that the healing process is satisfactory.

The above detailed treatment is that suitable for cases of uncomplicated ulcer, for ulcer preparatory to surgical measures, and for treatment after certain surgical procedures. The period of time for which it should be applied depends upon circumstances. Before operation it should be applied preparatory to such measures as gastro-jejunostomy and partial gastrectomy with or without pyloric occlusion. Perhaps the results of such operations would be improved and not so many anastomotic ulcers result if this were done. In the series under review quite a number of patients were "brought in off the waiting list" and operated upon the next day.

After such operations surely a period of medical treatment in bed, over and above the usual two weeks or so, would be of benefit to the patient and would give more chance for the undoubted good results likely to follow surgery.

The case presenting the unfortunate syndrome of the partially obstructed pylorus calls for special comment. As has been indicated before, this may be due to cicatricial stenosis, inflammation or sphincteric inefficiency or a combination of these factors. In any one case it is difficult to arrive at an exact conclusion as to which factor is mainly responsible. Therefore it is advisable to give the patient the benefit of the doubt and treat him medically, provided always that the vomiting and resulting starvation and dehydration are not such as to endanger his life. Under suitable treatment many such ulcers will clear up—they are those in which the causative factors are either inflammation or sphincteric inefficiency or both. If the condition of the patient is good, medical measures may have to be carried out for some weeks before the desired result is obtained. In actual practice the after-history of patients so treated is often unsatisfactory. Many of them suffer a relapse when they leave hospital, the main reason being that they cannot or will not carry out the proper after-treatment. Whether surgery offers better results is hard to say; for, if the patients do not carry out after-treatment after medical measures, they are not any the more likely to do so after surgical measures have been undertaken. Sippy states that of all cases of partial pyloric obstruction, 85% are due to non-cicatricial causes and 15% to scarring from invasion of the pyloric region by ulcer. In those cases that do not clear up by medical measures in a suitable period of time, surgery is indicated. In this regard surely it is bad medicine to "hang on" to a case too long, hoping for the best, and then hand over a half-starved, dehydrated "bad risk" to the surgeon. Again, it is another example of the principle that every ulcer case should be treated on its own merits.

The problem of the large gastric ulcer is different from that of the duodenal and smaller lesser curvature ulcer. Here the need for surgical treatment is surely greater. When the large size of the ulcer, surrounded as it is by an œdematous stomach, with the regional glands enlarged and unhealthy-looking, is considered, even the most optimistic physician must surely have his doubts as to the results of medical treatment. But here again a period of medical treatment should be carried out to prepare the field for the surgeon.

TABLE II.

Relative Efficiency of Alkalis in terms of Sodium Bicarbonate = 100.

Alkali.	Result.
Magnesium oxide	317
Magnesium peroxide	160
Magnesium carbonate (basic)	145
Sodium bicarbonate	100
Sodium citrate	61
Calcium phosphate (tribasic)	61
Potassium citrates	58
Magnesium phosphate (tribasic)	51
Calcium carbonate	20
Bismuth oxycarbonate	2

Hæmorrhage in Peptic Ulcer.

Hæmatemesis is a frequent symptom of peptic ulcer, while melena is an almost constant feature. In this series hæmatemesis occurred just before or just after admission on 83 occasions out of the 550

cases of ulcer. In addition a further group of 36 cases of probable ulcer presented this symptom. In every case the blood vomited was an appreciable amount, varying from a cupful to a "bucketful". Those cases with an indefinite history of "having vomited some blood" or "streaks of blood" have not been included. The bleedings after operations for ulcer, of which there were several examples, have also been excluded.

As Table III shows, in many cases the hæmatemesis was repeated. In addition, eleven cases of severe melena with no hæmatemesis occurred in this series (Table IV). The severity was judged by the hæmoglobin percentage on blood examination; those having readings of 35% or lower were regarded as "severe".

TABLE III.

Cases of Hæmatemesis in this Series.

Type of Ulcer.	Male.		Female.		Total.
	Single.	Repeated.	Single.	Repeated.	
Gastric ulcer	15	11	4	5	35
Duodenal ulcer	28	14	5	6	53
Stoma ulcer	1	3	—	1	5
Probable ulcer	14	9	9	4	36

TABLE IV.

Cases of Severe Melena in this Series.

Ulcer.	Male.	Female.
Duodenal ulcer	8	1
Peptic ulcer (? site)	2	—

The mortality in the 83 cases was five, that is, a mortality rate of about 6%. Of the eleven patients with melena, one died, giving a mortality rate of about 9%.

Similar figures obtained from other sources are of interest, for they would naturally tend to influence treatment of the condition. Stewart at Leeds found that in a series of *post mortem* examinations 0·41% of the total deaths was due to hæmorrhage from peptic ulcer.

Crohn in New York had 101 severe hæmorrhages in 426 cases of peptic ulcer. Of these, 94 patients were medically treated, with four deaths; seven were surgically treated, with five deaths.

Bulmer, from Birmingham, reports 578 patients with severe hæmatemesis, with 67 deaths, a mortality of 11·4% (series of 1902 to 1931).

Figures giving a mortality of as high as 25% have also been published.

Gordon Shaw, of Melbourne, states that of 330 patients with peptic ulcer, 37 had hæmorrhage. Of these, 35 were treated medically, with five deaths, two were operated upon and recovered.

The best line of treatment is still unsettled. Some advocate medical measures; others advise operation with a view to controlling the bleeding area, with or without a preliminary transfusion.

The medical treatment consists of complete rest in bed, and morphine given hypodermically helps to obtain this as well as to quieten down gastro-intestinal movements; the morphine is usually combined with atropine. With the exception of ice-chips and cold water, nothing is given by the mouth until the hæmorrhage has been arrested for some thirty-six hours or so. Fluids are given by the bowel in the form of glucose and saline solution.

Some authorities advocate washing out the stomach, for they maintain that the presence of blood clots in this organ stimulates peristalsis and acid secretion. The wash-out should be done with cold water, and when this is returned clear some adrenaline solution should be left in the stomach.

In addition to the above, in the case of severe hæmorrhage a blood transfusion should be carried out and repeated if necessary. The experience with the constant drip method of transfusion is as yet not extensive enough to indicate whether this offers any advantage over the more usual procedure. Hurst suggests that any patient with a hæmoglobin content below 40% requires a transfusion, and this may be taken as a guide. As soon as the hæmorrhage has been arrested for the thirty-six hours as indicated above, the ulcer *régime* is commenced. In the Adelaide series all the patients were treated medically, so I shall leave the discussion of surgical treatment to Dr. Ian Hamilton.

In the treatment of hæmorrhage in a case of chronic ulcer a point that has to be considered is whether the bleeding is coming from the chronic ulcer or whether it is coming from complicating acute ulcer or ulcers. As has been indicated before, the coexistence of chronic gastric or duodenal ulcer and acute ulcer or ulcers is not uncommon. Further, hæmorrhage is more commonly due to acute than to chronic ulceration. Many years ago Hale Winter recognized that hæmatemesis was often due to not one, but many bleeding areas, especially in the stomach; to this condition he gave the name gastrostaxis.

In the Adelaide series, on 36 occasions no radiological evidence of ulcer was found four weeks or so after the hæmatemesis occurred. As no other cause that would account for this was found, it may be presumed that most bleedings were due to acute ulceration. After all, with the exception of carcinoma of the stomach, the other diseases that may bring about hæmatemesis are relatively uncommon. In this series of 36 cases no deaths occurred. However, the fact remains that in any one case with severe hæmorrhage, even if it be known that a chronic ulcer exists, it is impossible to say whether the old or an acute ulcer is at fault. In considering the histories of the six fatal cases of this series there is one significant fact, however. On two occasions there was profuse loss of bright blood *per anum*, and this was observed in none of the other cases, totalling 138. Only one of these two came to autopsy, and then an eroded artery was found in a chronic ulcer. Surely the passage of bright blood *per anum* must mean that the bleeding

is very rapid and probably from an eroded vessel. It is suggested that this might be an indication for operative treatment to tie off the bleeding point. A synopsis of the six fatal cases of hæmorrhage is added.

CASE 7308/36.—A male patient, aged fifty-four years, gave a history of ulcer for many years. He had been discharged from hospital five weeks previously after a stay of two months. He had a history of melena and hæmatemesis on the day before that admission. He had been quite well since his discharge from hospital till the day previous to his second admission, when he noticed melena. He had melena again on the day of admission, and his hæmoglobin value was 50%. On the next day he passed another tarry stool. On the following day he vomited four ounces of blood. His hæmoglobin value was 34% and he was given a transfusion of twenty ounces of blood. He vomited blood again and had a rigor. Four days later he had two more hæmatemeses, of four and ten ounces respectively. He was given a transfusion of sixteen ounces of blood, and died. At autopsy a small gastric ulcer was found.

CASE 1796/32.—A male patient, aged fifty-nine years, gave a history of repeated hæmatemesis for five years. He had had gastro-enterostomy performed four years previously. He had never been free from symptoms. On the day previous to his admission to hospital he vomited two pints of blood. On the day of his admission he had great abdominal pain and passed bright blood *per anum*. His abdomen was rigid. Laparotomy was advised, but was refused. On the next day he was comfortable. On the next day he had another severe hæmorrhage and died. No transfusion was given.

CASE 5347/34.—A male patient, aged fifty-two years, gave a history of ulcer extending over the last twenty-three years. On admission to hospital he gave a history of recent hæmatemesis. On the evening of his admission to hospital he vomited blood and passed a tarry stool. On the next day he had a sudden collapse, vomited eleven ounces of blood and died. No transfusion was given.

CASE 4403/32.—A male patient, aged seventy-four years, gave a history of ulcer for one year. On the night previous to his admission to hospital he vomited "half a dipper" of blood. Six days after his admission he again vomited blood. Five days later more blood was vomited and he died. At autopsy a chronic duodenal ulcer, extending to surround the common bile duct, was found. No blood transfusion was given.

CASE 4392/35.—A male patient, aged fifty-two years, gave a history of ulcer for the last twenty years, worse during the last two months. On the day of his admission to hospital he vomited a pint of blood and passed dark clots *per anum*. Later he passed a pint of bright blood *per anum*. On the next day he was given a transfusion of eighteen ounces of blood and died forty-eight hours later. At autopsy a large chronic duodenal ulcer, which had eroded an artery, was found.

CASE 2799/32.—A male patient, aged forty-four years, gave a history of "peptic" ulcer for two years. Two weeks previously he had noticed melena and had become gradually weaker. On admission to hospital he was exhausted and pale. His pulse rate was 120 to 140. He was given a transfusion of twenty ounces of blood. He became unconscious for a while. On the next day he was a little better, but on the following day he was weaker and had a transfusion of sixteen ounces of blood, but died.

Perforation of Ulcer.

In this series perforation of an ulcer occurred on 117 occasions; of the patients, two died almost immediately after admission to hospital and so were not operated upon. The anatomical distribution of these ulcers is shown in Table V. It will be seen that perforation is relatively more common in the stomach than in the duodenum, and definitely more fatal. The combined mortality rate was 22·5%, the rate in gastric ulcer being 32·5% and that in duodenal ulcer 16%.

TABLE V.

Anatomical Distribution of Ulcers Perforating.

Type of Ulcer.	Cases.		Deaths.	
Pyloric	7		1	
Prepyloric	17	45	6	14
Lesser curvature	20		7	
Greater curvature	4		1	
Stomach	2		—	
Duodenum	62		10	
Anastomotic	3		1	

NOTE.—Two patients dying immediately after admission are not included. All ulcers except one occurred in men.

An interesting feature, previously pointed out by Hamilton Bailey, was that in quite an appreciable number of cases the pulse rate was not greater than 80 per minute. A rapid pulse is thus not a necessary feature for some six hours after perforation. Another feature was that many of the patients gave no history of indigestion, while nearly every ulcer at operation was recorded as being tough and obviously chronic. This is ample proof that an active chronic ulcer may exist without giving definite symptoms.

Further, no ulcer perforated while the patient was under ward treatment, and practically none perforated while the patients were carrying out ambulatory treatment. No record was found of perforation occurring in one of the medically treated cases of this series.

The treatment of this condition is immediate operation. The longer after the onset of acute symptoms this is done, the more grave does the prognosis become. In this series patients with a history of over twelve hours' illness often recovered.

Alkalosis.

The interesting and often unrecognized complication of the alkaline treatment of peptic ulcer known as alkalosis was first noted by Sippy in 1915. It is not recognized, first, because it is not thought of and, secondly, because its onset is usually insidious. The condition gradually develops, and the attendants, both medical and nursing, tend to regard the condition of the patient as being due to "nerves" or "touchiness" or "home-sickness", or something of the sort.

The alkalosis occurs more frequently when the soluble alkalis, such as bicarbonate of soda, are used, especially if they are those excreted by the kidney and not the bowel.

At one time it was thought that the syndrome occurred only in duodenal ulcer, and then usually with pyloric obstruction; but this is not so, for alkalosis in gastric ulcer and other conditions, such as diarrhœa and vomiting, has frequently been recorded. Its onset occurs during the first ten days of treatment, and most frequently during the middle of this period; but its onset may be delayed

for a year or more, provided treatment be continued. Some individuals appear to be more sensitive to alkali than others, and in them large doses are not necessary to bring about symptoms.

The main feature is the change in the personality of the individual, who becomes grumpy, touchy, querulous, unfriendly and even mildly persecuted. He may complain of nausea or actually vomit, while loss of appetite and an emphatic distaste for milk and all things milky and a craving for the more tasty foods are frequent. Such symptoms as headache, body aches, cramps, blurring of vision, giddiness, abdominal colic, and so on, are usual. An important feature is the presence of casts, albumin and even pus in the urine, now strongly alkaline; this is accompanied by an increased blood urea content and carbon dioxide combining power of the plasma. Objectively, a rapid pulse, slightly raised temperature, drowsiness, with a definitely sick-looking appearance, such as pallor, dry-coated tongue, blood-shot eyes, and a general air of dehydration, should clinch the diagnosis. If the condition is not recognized and dealt with, more serious events may follow. The usual sequence is stupor, convulsions, coma and death.

In this series no patient that could be definitely said to have had this condition was recorded. For some time it has been a routine to have a blood urea estimation at the end of the first week of treatment, and in no case was an abnormal reading found.

In my private practice I have seen two cases, one ending fatally because I did not then know of the existence of the condition, and in the other, occurring only recently, the patient quickly recovered when the alkali was reduced. It might be said of alkalosis: to be on the look-out for it is to recognize it; otherwise it creeps in unrecognized.

Finally, and perhaps the most important, a few words about the after-care of peptic ulcer, treated either medically or surgically. Even if the ulcer is healed or the ulcer-bearing area is removed, the underlying diathesis remains and so the tendency to produce further ulcers remains. As much as possible the individual should live at a lower speed and take plenty of time over meals and a rest before meals. To come in tired and bad-tempered from a day's work, in the winter especially, and immediately to bolt an unsuitable meal is to court trouble. A quarter of an hour in front of the fire and a peaceful, suitable meal are desirable.

The diet should be made up of those things that are not mechanically irritating to the alimentary mucosa and that do not stimulate excessive acid secretion. The taking of an alkaline powder after each main meal is a safeguard and a habit soon acquired. Any excess of alcohol or tobacco should be avoided, and areas of focal sepsis should be cleared up. Printed diet sheets showing what is allowable and what is not should be given in triplicate to the patient—one for himself, one for his cook and one in reserve. Such diet sheets—the bland diet number 4 of the London Hospital—are set out below. Morning tea, afternoon tea and supper are a help.

I inquired from my out-patient sister the other day why peptic ulcer was found more commonly in men than in women. She replied: "Well, they take longer over meals and take more between meals; and, sir, women are more even-tempered than you men." Perhaps she was right; I do not know!

Diet Table—Bland Diet Number 4 (London Hospital).

Foods and Drinks Allowed.

Milk and Eggs.—Milk prepared in any way—junket, custard and well-cooked milk puddings, milk soup; plain cream, made-up cream, ice cream, or cream soups; butter; eggs lightly cooked, beaten as eggs and milk, or as custard.

Fish and Meat.—Fish boiled, steamed or baked, but not fried; chicken or game, roasted or baked; tripe, brains or sweetbread, well cooked but not fried; meat—beef or mutton occasionally, roasted or baked, but not boiled or stewed.

Vegetables.—Potatoes well cooked or mashed; cauliflower, spinach, carrots, well cooked and rubbed through a sieve; tomatoes fresh or cooked if rubbed through a sieve.

Fruit.—If raw, avoid skins and pips; if cooked, always rub through a sieve; apricot meringue, red currant, apple or marmalade jelly, damson cheese.

Various.—White bread, cut thin and toasted dry; any kind of smooth plain biscuit; plain sponge cake or light madeira cake without fruit; jellies, plain chocolate, sugar, honey and golden syrup.

Drinking.—Drink sparingly with meals. You may take plenty of water between meals. Freshly made weak tea, especially China tea; coffee with milk; fruit juice; milk. A small quantity of light wine is allowed with dinner or supper.

Instructions as to Meals.—Take your meals regularly and avoid hurrying over them. Large meals are always bad; frequent small meals should be your aim.

Avoid taking a meal when you are tired or when you have cold hands or feet. Lie down, rest, and get warm first.

Eat and chew thoroughly.

Foods and Drink Forbidden.

Fried foods of all kinds are forbidden.

Soups and Meat Dishes.—Avoid meat soups and rich gravies, twice-cooked meats, sausages, made-up dishes, pork, "high" game, and all tough meat.

Fish.—Avoid salmon, sardines, dried fish.

Condiments.—Avoid spices, pepper, cayenne pepper, curries, vinegar relishes, pickles, chutney and mustard.

Vegetables.—Avoid cabbage, peas, beans, celery, onions, watercress and cucumber. Other vegetables must be passed through a sieve. You may add fresh butter to vegetables, but they are not to be cooked in fat. Avoid fried or chip potatoes.

Fruit.—Avoid all pips and skins of fruit, whether raw, cooked or in jams. Avoid raw apples or melon, marmalade jam, lemon peel, currants, raisins, figs, nuts and all unripe fruits.

Various.—Avoid rich pastry or puddings, new bread, brown bread, porridge and fruit cake. All cheese other than cream cheese is forbidden, and so is cooked cheese in whatever form.

Drinking.—Avoid cocktails, spirits. Never take alcohol on an empty stomach. Avoid strong tea, black coffee and cocoa.

Smoking.—Give up smoking if indigestion is present.

Acknowledgements.

I should like to thank the members of the staff of the Adelaide Hospital for allowing me to use the records of patients under their care. Also I should like to record the help I have had from the book "Gastric and Duodenal Ulcer", by Hurst and Stewart.

SOME REMARKS ON THE TREATMENT OF PEPTIC ULCER.[1]

By IAN HAMILTON, M.B., B.S., F.R.C.S., F.R.A.C.S.,
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You have just heard a very able thesis on some medical aspects of the cause, the life history, the diagnosis and the treatment of peptic ulcer, so much so that you probably have gained the impression that peptic ulcer is once again the clover patch of the physician; but I wish to tell you that chronic peptic ulcer, with certain well-defined reservations, is a disease which is essentially one for the care of the surgeon. The reservations which, as a surgeon, I put before you are these: (i) A peptic ulcer can be declared "chronic" only after it has been present for some months. Most have longer histories, but, as Dr. Beare has stated, some chronic ulcers manifest themselves first by rupturing or bleeding without a previous "ulcer history". (ii) Patients suffering from peptic ulcer rarely come under the surgeon's care until the ulcer is declared "chronic" and, unless it is complicated, until reasonably efficient and reasonably applicable medical treatment has failed to produce a progressive diminution in the size of the ulcer as shown by progressive X ray examination.

But since X ray examination frequently fails even to locate the ulcer correctly, it is rarely possible to show progressive diminution in its size. Therefore, for practical purposes I find it necessary to take the following signs as sufficient indication that medical treatment has failed: (i) the persistence of symptoms when the prescribed diet and powders are discontinued, or (ii) the persistence of any other signs, for example, X ray evidence that the ulcer is still present. The known habits and life history of peptic ulcer tend to show more and more plainly that few chronic ulcers, especially in working people, are cured without surgical assistance; and I believe that even if an ulcer thought to be chronic could be healed by medical treatment extending over some months, it would be better to aid that medical treatment by simple surgery where this was possible. In really skilled surgery the mortality of simple ulcers should be slight, and surgery provides a method of localization far superior to any other and enables the living pathology of that particular ulcer to be accurately studied, whether the ulcer is on the stomach or on the duodenum, is attached or free, is simple or malignant. I am aware that many physicians will say that a large proportion of ulcers can be cured by medical treatment alone; but Dr. Beare finds that out of a very large number of patients suffering from ulcers, few are ever well enough to be finally discharged from the Adelaide Hospital, and few ever leave the hospital before they die, unless they become financial, and so are ineligible for treatment there.

Admittedly, my statistical records of 150 patients who were operated upon at the Adelaide Hospital (see Tables I and II) would not encourage the physician to employ the surgeon if he could avoid it; but I hold that the result can be tremendously

TABLE I.
Analysis of 150 Adelaide Hospital Cases in which Operation was Performed.

Type of Case.	Number of Cases.	Deaths.	Percentage of Deaths.
Ruptured ulcer	74	12	16·2
Deliberate operation performed	71	11	15·5

TABLE II.
Analysis of Cases: Gastric and Duodenal.

Type.	Ruptured.		Not Ruptured.	
	Number of Cases.	Deaths.	Number of Cases.	Deaths.
Gastric	36	8	39	7
Duodenal	38	4	32	4

improved by having every patient medically treated for one or two weeks before operation. There should be a careful discussion as to the type of operation most suitable to the patient in question, both before the operation is attempted and while the operation is being performed. All pre-operative information and information gained by exploration should be utilized by the surgeon, the physician and the radiologist in the operating theatre; and to this information should be applied, first, a knowledge of the possibilities of surgery in any particular case; secondly, a meticulous regard for surgical technique, such as is acquired only by skilled operators who are always open to suggestion and who constantly criticize themselves, who accept criticisms from their collaborators, and who check up their results; thirdly, a meticulous *régime* of post-operative medical treatment should be instituted. Results will never be first class until these desiderata and perhaps others are fully satisfied. Faults may occur in the method of stitching, in the space between the stitches, in the amount of stomach excluded or excised, in the size, the position and the angle of the stoma, in the length of the proximal loop, and in the possible rotation of the loop, just to take a few instances.

What operations of proved value are to be considered? This question must be discussed according to the ulcer to be treated. It can be laid down as a broad principle that it is unsafe to

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 29, 1937.

attempt the excision of duodenal ulcers, and that gastric ulcers must be excised, whatever else is done. There are conceivable exceptions to each rule.

Duodenal Ulcer.

The outstanding operation for duodenal ulcer has until lately been gastro-enterostomy, but Devine has recently published an excellent paper giving some important directions as to the best method to employ in the surgical treatment of duodenal ulcer. Of those patients who were treated in the past by gastro-enterostomy alone, only two-thirds were permanently freed from symptoms, leaving one-third in whom the result was unsatisfactory; and we have to seek the reason for this. Broadly, the faults may be classified as follows.

Wrong Diagnosis.—In quite a considerable number of cases of supposed duodenal ulcer the diagnosis was not properly verified at operation; but nevertheless, as nothing else was found to account for the symptoms, gastro-enterostomy was done with the certain result that the follow-up showed a failure or worse. The diagnosis should be verified and agreed upon by both the skilled surgeon and his skilled assistant. On the other hand, further conditions may develop to cloud the issue, for example, pregnancy.

Improper Selection of Cases for Gastro-Enterostomy.—Patients with a high hyperchlorhydria will almost inevitably develop a gastro-jejunal ulcer after gastro-enterostomy unless something more than a gastro-enterostomy is done. No patient should ever be subjected to operation without a test meal to determine the degree of the hyperchlorhydria.

Failure in Technique.—Gastro-jejunostomy is very susceptible to even slight errors in technique. It is hardly necessary to mention that unabsorbable sutures will almost inevitably cause gastro-jejunal ulceration. The stoma may be too small or too large, it may be to high or too low, the proximal loop may be too short or too long, or other ancillary pathological conditions in the abdomen, for example, chronic appendicitis, may not be recognized and dealt with.

Insufficient or Imperfect Pre-Operative and Post-Operative Medical Treatment.—Many authorities consider that the strict post-operative medical *régime* should be continued for six months. Alcohol and tobacco should be very strictly limited or entirely prohibited during this period.

The following is my own post-operative gastric diet; it has been modified from the West London Hospital diet:

First twelve hours: Mouth washes only; rectal saline injections four-hourly.

First day: Two ounces two-hourly—albumen water, orange juice, raisin tea, diluted milk or citrated milk, barley water, rice water; rectal saline injections four-hourly.

Second day: Three ounces two hourly as above.

Third day: Four ounces three-hourly; rectal saline injections six-hourly.

Fourth day: Five ounces three-hourly; water as desired; add calves-foot jelly, Allenbury's sweet whey, Valentine's meat juice, arrowroot.

Fifth day to eighth day: Start on treatment with alkaline powder; water as desired. 7 a.m., powder; 8 a.m., creamy milk (three ounces), or typhoid bread and milk, or semolina or arrowroot, and cup of weak tea; 9 a.m., powder; 10.30 a.m., creamy milk (three ounces), or Benger's food (three ounces) or arrowroot; 12 noon, baked custard, creamy milk; 12.30 p.m., powder; 4 p.m., creamy milk (five ounces) or Benger's food, flavoured if desired, with coffee or tea; 6 p.m., milk as above, thin bread and butter, tea; 7 p.m., powder.

Eighth day to fourteenth day: As above, but add poached egg for breakfast, with bread and butter, any lightly cooked egg, well-cooked rice, Benger's food; pounded fish for lunch.

From fourteenth day for three weeks follow the same plan as regards powders and meals, but may take any of the following suitable foods in addition for breakfast, dinner or tea: steamed cod, snapper or whiting; meat without gristle, fat or gravy; chicken, turkey; lamb without fat, once daily; all well-cooked vegetables except cabbage, cauliflower, brussels sprouts or turnips; milk, eggs, butter, bread.

Avoid salt, extractives, meat soup, broth, condiments, spices, pickles, alcohol, skins and seeds of fruits and vegetables, salads.

After the second day of treatment, if not vomiting, take liquid paraffin three times a day for the bowels.

Other Factors.—There are patients in whom the ulcer-forming habit is incorrigible. One such came into the Adelaide Hospital out-patient department some months ago and I was asked to see him. He had had four ruptured ulcers. Some patients develop a neurosis and cannot be cured, while some on "relief" may possibly be malingering.

What more than a gastro-jejunostomy can be done for a duodenal ulcer? I have previously stated that excision is unsafe, and it is unsafe because the duodenal stump after partial duodenectomy leaks in a large proportion of patients, and this usually gives rise to a fatal peritonitis. But two types of patients, namely, young subjects and those with a high acid curve, must have something more than a gastro-jejunostomy done to reduce the acid formation and so prevent jejunal ulcer from following. Partial gastrectomy by one of the several methods will effectively do this, but the experiments of Devine show that what is now termed gastric exclusion, with or without excision of a part of the acid-forming area of the stomach, gives excellent results in those difficult cases with less risk than the ordinary gastrectomy. In this operation the stomach is divided across somewhere about its middle, according to the judgement of the surgeon. The proximal part is anastomosed to the jejunum and the distal end is closed with or without removal of some of it. The gastric juices from the proximal part go on into the jejunum with the food, while the food secretions from the distal portion pass through the pylorus into the duodenum; digestion is only slightly interfered with, and the discomforts of removal of the stomach are avoided.

In dealing surgically with duodenal ulcer, the operating surgeon must have in his repertoire of operations a number of procedures, such as posterior and anterior gastro-enterostomy, gastro-duodenostomy, excision alone with the cautery or knife,

excision combined with gastro-enterostomy or reconstruction of the pyloric outlet, pyloric occlusion with gastro-enterostomy, partial duodenectomy combined with partial gastrectomy by the Billroth I or Billroth II type of anastomosis, antral exclusion, jejunostomy *et cetera*; but it is not possible to spend time on all these tonight.

The Surgical Treatment of Gastric Ulcer.

This subject is so vast that in this discussion I cannot do more than generalize. The experience of the Mayo Clinic is such that Balfour remarks that "it is known that certain small gastric ulcers may heal under intensive medical management and that recurrence can be prevented in a considerable percentage of cases". I must therefore reiterate my previous statement that chronic gastric ulcer is a surgical disease, and more forcibly because by surgery and surgery alone can we determine at a sufficiently early stage whether the ulcer is malignant or not. Moreover, gastric ulcer and its associated gastritis become progressively less curable with delay in the application of surgery. If care is exercised in the selection of patients for operation, if the selection of the operative procedure to suit the case is made with minute care, and if meticulous technical skill is used in operating, almost 100% of patients will be cured of their gastric ulcers by surgery. The ideal to be aimed at in the surgery of gastric ulcer is complete removal of the lesion with provision for good drainage of the stomach by the safest operation which will permanently change the motor and secretory function. A number of operations are in use: (i) excision alone, (ii) gastro-enterostomy alone, (iii) excision and gastro-enterostomy done with cautery or knife, (iv) excision combined with reconstruction of the pyloric canal, (v) segmental or sleeve resection, (vi) partial gastric exclusion (Devine's operation), (vii) partial gastrectomy, (viii) jejunostomy. Unquestionably almost all authorities agree that the operation of partial gastrectomy is the operation chosen by competent surgeons for all large gastric ulcers if it can be performed with reasonable safety. Some lesions can be treated by one or other of the other methods, such as gastro-enterostomy alone, which may be used for patients who are "poor risks" and who have large or attached ulcers; jejunostomy is especially indicated as a "building measure" in these cases. Excision alone is almost never used, as it does not provide for drainage of the stomach and alteration of its function. Excision combined with gastro-enterostomy is an excellent operation and one frequently employed by Walton. It provides for all the essentials, but my experience of it is that it is possibly more difficult to manage well, from a technical point of view, than is partial gastrectomy. Kirscher and Phillipides have recently published statistics tending to give more encouragement than ever before to the use of the operation of partial gastric exclusion in suitable cases of both gastric and duodenal ulcer. In Tables III and IV are found figures from Balfour dealing with this question.

TABLE III. (After Balfour.)

Showing Frequency with which Various Operative Procedures are Employed in the Treatment of Gastric Ulcer, 1928 to 1931, Mayo Clinic.

Operative Procedure.	Frequency Percentage.
Gastro-enterostomy: Anterior, posterior, with or without enteroanastomosis	17·6
Excision alone: Cautery, knife, wedge, segmental	8·8
Excision and gastro-enterostomy	26·1
Excision and reconstruction of the pyloric canal	6·6
Partial gastrectomy: Billroth I, 16·0%; Billroth II, 7·6%; posterior end to side anastomosis, 72·0%; anterior end to side anastomosis, 3·4%	38·0
Miscellaneous: Partial gastric exclusion, jejunostomy, total gastrectomy, and so forth	3·7

TABLE IV. (After Balfour).

Showing Mortality in a Series of 2,244 Consecutive Cases in which Operation was Performed because of Acute, Subacute or Chronic Gastric Ulcer, 1906-1927.

Operation.	Cases.	Deaths.	Percentage.
Partial gastrectomy, all types	502	27	5·4
Knife or cautery excision, with or without gastro-enterostomy, gastro-duodenostomy or pyloroplasty, and so forth	1,202	46	3·8
Gastro-enterostomy, posterior or anterior	540	21	3·9
Total	2,244	94	4·1

Treatment in Gastric and Duodenal Bleeding.

Provided that the clinical features and X ray investigation of the condition lead to a diagnosis of peptic ulcer, then the question of surgical intervention in those cases in which bleeding occurs will always arise, and a search of the literature helps to indicate the best treatment. Rule of thumb cannot help; individual consideration of individual cases is the keynote.

Acute ulcers, if they can be diagnosed, always yield to medical treatment. In chronic ulcers, with repeated slight to moderate hæmorrhage, the indication is that the ulcer is active. Experience shows that medical treatment usually controls the bleeding, and the ulcer should be treated intensively by medical means. If the usual indications for surgery are present, then deliberate operation should be performed after the building up of the patient. Should his hæmoglobin value be low, blood transfusion should be given. The ulcer, of course, must be excised whatever else is done.

Massive Hæmorrhage.—Massive hæmorrhage usually occurs by erosion into one of the great vessels, such as the right gastric artery, the left gastric artery, the gastro-duodenal artery or the splenic artery, and may be rapidly fatal. It is arrested by a plug of fibrin forming as the blood pressure falls, and once a great vessel is eroded hæmorrhage is extremely likely to be recurrent. Operation without doubt should be carried out in these cases and the ulcer itself must be attacked; but when should the operation be done? The mortality associated with immediate operation at the London Hospital was found to be 38%. However, Pannett has suggested that immediate operation with synchronous blood transfusion would enable

operation to be carried out without a heavy mortality. So long as the bleeding is stopped, further operation can be performed in two or three weeks; but most authorities agree that if these patients survive their acute hæmorrhage, operation should be done two or three weeks later, so as to avoid a recurrence.

Statistics of the Adelaide Hospital.

The statistics of the Adelaide Hospital for operations on patients with gastric and duodenal ulcer are set out in Table V.

TABLE V.
Operations Performed.

Gastric Ulcer.		Duodenal Ulcer	
Closed: 30 (5 deaths).	Gastro-enterostomy: 18 (2 deaths).	Closed: 31 (2 deaths).	Pyloroplasty: 1.
Gastro-enterostomy and closure: 4 (1 death).	Gastro-gastrostomy: 1.	Closed and gastro-enterostomy: 6.	Occlusion of pylorus and gastro-jejunostomy: 1.
Nothing done to ulcer: 1 (1 death).	Excision, pyloroplasty and gastro-enterostomy: 1.	Excision and pyloroplasty: 1 (death).	"Repair": 1.
	Cauterization: 1 (death a week later from rupture).		Gastro-enterostomy: 19 (4 deaths).
	Oversewn: 1.		Gastric exclusion: 0.
	Excision and gastro-enterostomy: 7 (2 deaths).		Gastrectomy: 4.
	Gastrectomy: 7 (1 death).		"Scar oversewn": 1.
	Excision: 3 (1 death).		Excision: 1.
	Gastric exclusion: 0.		Pyloroplasty and gastro-jejunostomy: 1.
			Invagination of ulcer and gastro-jejunostomy: 2 (1 death).
			Pylorectomy: 1.

Causes of Death in the Present Series.

The following is an analysis of the causes of death in the present series.

Cases of Ruptured Ulcer.

1. The ulcer was situated at the pyloric end of the stomach; it was oversewn. The patient died of peritonitis four days after operation.

2. The ulcer was on the greater curvature; it was oversewn. The patient died of empyema one month later.

3. The ulcer was on the posterior wall of the stomach; it was oversewn. The patient died of shock on the same night.

4. The ulcer was on the middle of the lesser curvature; it was found at the *post mortem* examination, not at the operation. The patient died of peritonitis three days after his admission to hospital.

5. The ulcer was on the first part of the duodenum; it was excised and pyloroplasty was performed. The patient died of melena eleven days after operation. At the *post mortem* examination two more ulcers were found in the duodenum.

6. The ulcer was on the anterior wall of the duodenum; it was repaired. The patient died of peritonitis eight days after operation.

7. The ulcer was on the proximal part of the duodenum; it was repaired. The patient died of peritonitis four days later.

8. The ulcer was on the lesser curvature; it was repaired. The patient died of shock next day.

9. The ulcer was on the lesser curvature; it was repaired. The patient died of subphrenic abscess which was drained twenty-eight days later.

10. The ulcer was on the first part of the duodenum; it was repaired. The patient died of subphrenic abscess and bronchopneumonia one month later.

11. The ulcer was on the lesser curvature; it was repaired and gastro-jejunostomy was done. The patient died of intestinal obstruction sixteen days later.

12. The ulcer was on the lesser curvature; it was repaired. The patient died on his return to the ward.

Deliberate Operations.

1. A duodenal ulcer and hæmatemesis occurred in a patient with a high acid curve. He died of peritonitis two days after gastro-enterostomy had been performed.

2. A moderately large duodenal ulcer was present. The patient was given no test meal. Gastro-enterostomy was performed. The patient had to have a second operation, when it was found that the sutures had given way and that there was a volvulus of the small intestine. The patient died of intestinal obstruction and pneumonia nine days after the first operation.

3. The ulcer was not felt, but the patient had pyloric stenosis. Gastro-enterostomy was performed. The patient died of cardiac failure nine days after operation.

4. The ulcer was situated high on the lesser curvature, adherent to the pancreas. It was cauterized and repaired, and gastro-enterostomy was performed. The patient had to be operated on again on account of a volvulus of the proximal part of the jejunum, which was straightened out but recurred, necessitating a third operation. The patient died one month after operation.

5. The ulcer was on the lesser curvature. It was excised and gastro-enterostomy was performed. The patient, who was a female aged sixty years, died of myocardial failure two days after operation. *Post mortem* no abnormality was detected.

6. A gastric ulcer was present. Cauterization and gastro-jejunostomy were performed. The patient died of ileus two days later.

7. A large ulcer was present on the lesser curvature. Partial gastrectomy was performed. The

patient had been subjected to duodeno-jejunostomy two years before without relief. The pancreas was inadvertently cut in this operation and the patient died of acute peritonitis in one week.

8. The ulcer was on the posterior surface of the stomach. Gastro-enterostomy was performed. The patient died of partial obstruction of the intestines five days later.

9. A large ulcer was on the lesser curvature, adherent to the pancreas. Gastro-enterostomy was performed. The patient died of hæmatemesis from an eroded artery in the base of the ulcer two days later.

10. A duodenal ulcer was present. Gastro-enterostomy was performed. The patient died from pneumonia nine days later.

11. The ulcer was on the lesser curvature, adherent to the pancreas. It was excised, but the patient died of shock the following day.

MEDICO-LEGAL EVIDENCE AND AUTOPSIES.

By D. S. McKENZIE, D.S.O., M.B., Ch.M.,
Police Surgeon, Perth.

THE medical man required to testify as a witness in courts of law will usually find himself fulfilling two functions, namely, reporting bare facts and interpreting such facts to the best of his knowledge and ability. In the first rôle he does not differ materially from the ordinary witness, except in so far as he is expected, by virtue of his special qualifications, to be a more accurate observer of such facts as are of value in forming medical opinions; in his second rôle he acts as a member of a special class, commonly called experts, who, by reason of their training, knowledge and skill in a particular subject, are entitled to express opinions on facts observed by or presented to them—a position that the ordinary witness is not allowed to assume.

The medical man called in by the law in medico-legal cases should, if possible, have a wide experience; he should be able to elucidate the medico-legal questions as they arise; and he must be prepared to make a careful selection of medical facts and apply such medical principles as may be necessary to place the facts before the judge in a clear and non-technical light, so that the judge may instruct the jury clearly on those medical facts. But it must be remembered that any member of the medical profession is liable on occasion to be called as a witness in court, and that all that the court expects from him on such occasions is a fair average knowledge of those facts that come under his notice and are within his province as a medical man. It hardly needs emphasizing that he should not be in a hurry to pose as a specialist unless he is exceptionally well versed in the particular specialty. The specialist stands in the same relation to the rank and file of the profession as the High Court judge does to the ordinary barrister.

The duties of the medical jurist are quite distinct from those of the practising physician or surgeon. The latter is concerned with the treatment of disease and the saving of life; the former is an assistant to the law, for his specialized knowledge and expert observation aid not only in identifying the perpetrator of a crime, but also in protecting an innocent person from the results of a crime wrongly imputed to him. The medico-legal aspect of medicine will come within the duty of every doctor sooner or later, and it is well for medical practitioners to keep Taylor's "Medical Jurisprudence" handy and to browse within its covers from time to time. It should be remembered that the proceedings of the courts are public property; and if a medical man appears ridiculous or careless or ill-informed, such a happening is likely to react on his private work.

The first duty of the medical practitioner is to cultivate the faculty of accurate observation; the second is to have some general knowledge of the law of evidence. A Scottish judge remarked during the course of a murder trial that "a medical man, when he sees a dead body, should notice everything". The clothing of the deceased should be carefully examined on the spot, as well as the body, while a close scrutiny of the body as regards warmth and rigidity will be of help when the awkward question, "How long has he (or she) been dead?" is asked. Counsel for the defence invariably tries to find out what the medical man has omitted to observe. He makes as much capital out of sins of omission as he can, and he may put these before the jury so strongly that the medical evidence is liable to be discounted and the accused to be given the benefit of a doubt which does not really exist.

It is essential that evidence should be given in such an impartial way that a charge of bias cannot be directed against the medical witness. His findings must not be inspired by the police or by counsel. He should carefully consider them and stick to them unless further information raises substantial doubt. Should he be proved incorrect on some small point of opinion, he should admit it and not try to carry it off by bluff. Both the prosecuting and the defending counsel, and the judge as well, have probably been well informed of all the medical possibilities. The taking of full, clear and copious notes should never be neglected, for it might happen that weeks or even months elapse before the case is tried. Memory cannot be trusted, and it may be tested out by counsel in some cunning way peculiar to the legal profession. The law in regard to the admissibility of notes is very strict, and to render notes and memoranda admissible it is essential that they be made at the time of the case or as soon thereafter as possible. Though these notes cannot be read as evidence, but are only to refresh the memory, the judge may accept them in certain cases if requested to do so, and if no objection is raised by the defence. Time, place and date should be very carefully noted, and the time should always be given to the nearest clock figures.

When the medical practitioner is called on to do an autopsy he should take nothing for granted—the most obvious cause of death may be only secondary. In his report the plainest possible language should be employed, and exaggerated terms should be avoided. When the use of technical and anatomical terms is inevitable, their meaning should be given in parentheses. Coroners are not as a rule conversant with abstruse medical terms, and even judges can assume an air of ignorance when they feel so inclined. For example, the *pia mater* might be the name of the medical witness's patron saint for all they know, whereas if they were told that the covering of the brain was destroyed the meaning would at once be clear.

In stating the cause of death care should be taken that deductions and conclusions are based on facts of medical import only and on what the medical witness has himself observed. It should not be forgotten that copies of his report and depositions (that is, the written record of his oral evidence before either a magistrate or a coroner) are placed in the hands of the judge and counsel and are compared word for word with the evidence he gives at the higher court. Discrepancies between these depositions and later evidence give rise to very awkward questions when the medical witness is under cross-examination. Counsel for the defence emphasizes these differences to the jury and may suggest that little credence can be given to the evidence of a man who gives two different versions of the same transaction.

With regard to the law of evidence, a medical man in the witness box may undergo examination-in-chief, cross-examination, reexamination, and questioning by the judge or juror. The examination-in-chief is carried out by counsel in accordance with procedure, and the questions asked do not suggest their answers. Leading questions may be asked should the judge decide that material evidence is being withheld or if the witness's answers differ materially from his depositions, in which event he can be treated as a hostile witness.

In cross-examinations counsel seeks to get from the witness any facts that he thinks would aid his client. He is permitted to ask leading questions. This is the time in which questions are asked as to the witness's experience and qualifications; and other questions also are asked, some of a most irritating type. Avoid getting excited or angry, or attempting to attack counsel, for if he is getting the worst of the wordy battle he will plead for protection and the witness will receive what he is apt to consider an unjust rebuke from the judge. It is the duty of an advocate, if his client is submitting a version of the facts or an expert opinion which differs from that of the medical witness, to put it in substance to him so that it may be explained, reinterpreted or contradicted flatly; and if he adopts the position of an expert, the practitioner's qualifications as compared with those of others may reasonably be called into question. But all courts can, and indeed should, forbid questions which are intended to insult or annoy, or are needlessly offensive in the form in which they are asked, and also the witness is entitled to plead for protection from such questions.

Straightforward answers must be given. Evasions only get the witness into trouble. Lawyers win their cases on evasions. If he is not sure of an answer, the witness should say that he does not know, and stick to it.

Reexamination is carried out by the counsel who has called the medical witness. This reexamination is confined to matter which has arisen out of cross-examination, and clears up any points made obscure by the cross-examination. If at any time the witness cannot give a direct answer to a question without qualifying it, he must not hesitate to say so. The judge may ask any questions. Questions by jurors are at the discretion of the judge. It has been remarked before that counsel has certain privileges, so the witness should not get too angry if doubt is thrown on his ability or knowledge. Care must be taken when he is answering questions and opinions quoted from authorities. He should ask that the context be read; and if he disagrees, he should state his reasons.

With regard to professional secrecy, no special privilege is granted to a medical man when in the witness box. Any information obtained by him as a medical man must be given if asked for in the court. If he feels that his position in giving such information should be clarified, there is no objection to his asking the presiding judge whether he is compelled by law to answer, and the judge will give his ruling.

The Medico-Legal Post Mortem Autopsy.

It is never too late to perform a *post mortem* examination. No matter how long it is since death has occurred, there is always a possibility of finding some item of evidence and thus satisfying the ends of justice. Bones, hair and teeth are practically indestructible by ordinary decomposition. Some organs, such as the non-gravid uterus, are slow to decay. Arsenic, a common agent in poisoning cases, inhibits decomposition. No incomplete examination must satisfy the medical man; abdomen, chest and cranial cavity must be carefully examined. Notes must be made at the time of anything and everything observed. It does not matter if the notes are dirty or blood-stained; soiled notes are at least evidence of authenticity.

For a *post mortem* examination the following instruments and accessories will be required: two good strong knives (butcher's knives are excellent); a saw (again of the butcher's type); two pairs of scissors, one blunt-ended and the other sharp; a large dissecting forceps; a bone forceps; a couple of chisels and a hammer or mallet; a sail needle and coarse thread; two pairs of gloves; iodine; an overall; a steel tape; three or four clean, wide-mouthed bottles with screw tops; string; paper labels and a good supply of sealing wax. If the autopsy is performed in the country, a camera is very useful. A good supply of a strong deodorant,

such as "Sanitas" or "Solyptol", is useful. A bottle of whisky to dose up the police assistants is almost a necessity.

Having got the body on to a convenient table or trestle, and in a good light, the medical practitioner should note the main identification points: sex, apparent age, height, colour of skin and hair, deformities, wounds, tattoo marks, condition of teeth, and finally, dental plates. These must be carefully labelled and handed over to the police. (It will be recollected that in the "Snowy Rowles" case some dental work was identified by a New Zealand dentist and thus assisted in the identification of the burnt remains.) The race and any occupational marks should be noted, particularly any stains, dirt or mud on the clothing, boots or person of the deceased. Any hypostatic congestion should be observed and distinguished from bruising; *rigor mortis* and the degreee of putrefaction present should be noted. In recently dead bodies the temperature should be taken in the rectum and compared with the temperature of the outside of the body. This may give valuable aid in fixing the time of death. The condition of the orifices of the body should be noted; excoriations, burns, bruises and the presence or absence of any discharge should be looked for. Negative findings must be carefully recorded.

Very careful notes of the external inspection of the body must be taken, especially of marks of violence, no matter how trivial. The autopsy should be made on a routine, say, from the head downwards. In making the report it is suggested that notes on the external examination should be divided into two main groups: (*a*) marks of injuries not of themselves fatal, and (*b*) the more serious and possibly fatal injuries. In the first division, burns, even trivial, powder stains, abrasions, cuts, fingernail scratches, erosions and excoriations (especially those around the natural orifices), all the bruises and their size, should be noted. In the second group, fractures and dislocations, cut throat and other deep cuts, extensive burns, stabs and punctured wounds, gunshot wounds must be carefully noted, with their size and their exact position. Measurements should be given in inches, not in centimetres. Laymen are not conversant with scientific standards, and explanations are never very enlightening. It must be remembered that *ante mortem* gangrene is easily distinguished from putrefaction by the fact that *post mortem* putrefaction never produces pus.

The internal examination should be begun at the cranial cavity, because cutting of the jugulars and the innominate may drain the cerebral sinuses. In medico-legal *post mortem* examinations, when the brain is being cut, the portions must not be completely separated from the brain stem, since it may be necessary to reconstruct the brain and harden it for exhibition. The membranes should always be stripped from the base, and careful examination for basal fracture should be made. Brain injuries should not be probed; they should be traced by dissection.

In cases of suspected volatile poisoning from alcohol, carbon dioxide or hydrocyanic acid, a good specimen of blood should be taken from the heart cavity. In suspected air embolus the auricle should be carefully opened and any frothy appearance noted. The presence or absence of food in the stomach must not be neglected. If any is present, its character and smell should be noted. There will always be some food in the stomach for two hours after a meal.

Specimens should be taken in sufficient quantities. The analyst likes a fair amount of material to work on. They should be placed in the jars, and wrapped, sealed and labelled very carefully. These specimens and a tabulated statement must be handed to the police officer in charge of the case. A note of the officer's name and rank should be made.

In conclusion, it is well when the examination is completed to take pains to obliterate the marks as far as possible and to clean up the body so that relatives' feelings are spared. Whenever possible the *post mortem* examination should be postponed until after the body has been identified by relatives or friends.

Reports of Cases.

GASTRO-JEJUNOSTOMY AND RUPTURED PEPTIC ULCER.

By T. M. FURBER, M.B., Ch.M., F.R.A.C.S.,
Sydney.

GASTRO-JEJUNOSTOMY has been strongly urged, and as roundly condemned, as a routine addition to the treatment of ruptured ulcers; while some intrepid souls, mainly of middle European origin, have sunk the offending ulcer without trace by doing a partial gastrectomy. The position has with most people clarified itself; and few now increase the dangers of a man already in parlous straits by doing a gastro-jejunostomy unless there is some definite indication, the most generally recognized reason being that the stitches closing the leak have caused some stenosis.

In the following two cases it has seemed to the writer that he should perform an anastomosis for another reason, namely, to provide a safety valve through which intragastric tension could be relieved, in order to protect stitching which, on account of the friability of the tissues, did not seem trustworthy.

Some six years ago a man, sixty-four years of age, was in the Royal Prince Alfred Hospital on account of digestive trouble, which, after thorough investigation, was thought to be due to a carcinoma high up on the lesser curvature of the stomach. While he was still in hospital a perforation occurred, and after a laparotomy four hours later a ragged hole was seen in the stomach, roughly in the middle of an infiltrated and nodular area the size of the palm of the hand. The leak was stopped but imperfectly, so the left lobe of the liver was tacked over it and a posterior gastro-jejunostomy was performed, so that any rise in the tension in the stomach would be quickly relieved and would be less liable to disturb the precarious hold of the stitches closing the hole.

To the surprise and joy of everybody concerned the old gentleman recovered and walked out of hospital. It would therefore appear that the ulcer was simple, as it is impossible to believe that a hole in a carcinoma would heal.

Towards the end of 1936 a man, aged forty-five years, came from the country to place himself under the care of Dr. C. C. Finlay on account of digestive symptoms, which, considered in conjunction with his colour, his loss of weight, his achlorhydria, and an X ray report of a large filling defect on the lesser curvature of the stomach, had been thought to be due to a gastric carcinoma causing pyloric stenosis. Dr. Allan Walker concurred in the diagnosis and advised a laparotomy, with a view to performing gastro-jejunostomy. While this advice was being considered, the patient, who was still in hospital, was seized with agonizing pain, which was immediately recognized by Dr. Finlay as due to a perforation. Dr. Finlay then asked the writer to see him. As it had been so successful in the first case, laparotomy was advised, and the advice was accepted; and so with very mixed feelings the writer embarked on the adventure four hours after the catastrophe had occurred.

However, the gods were kind, and success rewarded a patient attempt to close two holes in infiltrated friable tissue, out of which, with disheartening regularity, the stitches tore. The holes were cobbled up somehow and a little comfort was extracted from the thought that the left lobe of the liver would cover the danger area and might become adherent if only the stitches held for a few hours. A posterior gastro-enterostomy was quickly performed and the abdomen was closed. It was felt that, although the forlorn-hope attack had a fair chance of success, it was likely at best to be but a Pyrrhic victory, as it was still thought likely that the trouble was carcinomatous.

The patient triumphantly falsified the gloomy prognosis, and within a few days began to eat and thrive, and some seven months later had gained two and a half stone. He felt and looked so well when recently seen that the question of giving an opaque meal to estimate his progress was not raised. It is impossible to be sure that the ulcer was simple, as to have removed a piece for examination would have added to the surgeon's already great troubles, but it is surely safe to say that the condition was not carcinomatous.

The cases seem worth a card in the index of memory for two reasons: first, that in the same way that a temporary caecostomy will help to prevent leakage after a resection of the large bowel, a gastro-jejunostomy will protect doubtful stitching, which otherwise might be expected to drag like an anchor in bad-holding ground; and secondly, that, as was advised by Sir Hugh Devine some years ago, all patients diagnosed as suffering from carcinoma of the stomach should, if secondary deposits are not found, be subjected to laparotomy, which might reveal a mistake in diagnosis and show that the condition was amenable to surgical treatment.

Reviews.

ANÆSTHETIC COMPLICATIONS.

C. J. M. DAWKINS, in his thesis on anæsthetic complications and their relation to basal narcosis,[1] reaches some arresting and significant conclusions from a careful analysis of over 16,000 inductions of anæsthesia, covering a period of fifteen years and including a wide variety of methods and agents. He compares particularly two distinct periods, namely, 1921 to 1925 and 1931 to 1935, representing the situation before and after the introduction of basal narcosis. He shows that substantial reduction in complications has resulted from the better methods of anæsthesia developed in recent years, but that the improvement has been largely offset by the unfavourable effects of indiscriminate basal narcosis. This has had an extensive application as a preliminary to general and local anæsthesia, but, despite its many obvious advantages, certain doubts about it have been accumulating in the minds of surgeons and anæsthetists, and this survey crystallizes them in no uncertain manner. That undue deference should be accorded to popular demand in this regard is to be deprecated; apart from this, a close study of Dawkins's findings will amply repay all concerned.

The outstanding feature is that with basal narcosis pulmonary morbidity was almost doubled and mortality from pulmonary complications definitely increased. The author has used the barbiturates almost exclusively for this purpose, and unfortunately makes very little reference to dosages. While he restricts the scope of his analysis, this also possibly accounts in some measure for the alarmingly high incidence of pulmonary complications, which are commonly associated with barbiturate poisoning. Better figures might have been obtained with a wider range of narcotic agents, but nevertheless, the necessity for minimizing abuse of physiological processes cannot be ignored.

An excellent review of the numerous factors which, either singly or combined, may operate in the production of such complications is given, especial emphasis being placed on the high incidence when upper abdominal surgery and basal narcosis are associated. The author appears to be amply justified in his conclusions that basal narcosis has impeded the reduction of anæsthetic complications, and that it should not be employed unless absolutely necessary.

The presentation of the subject matter is most concise, and the various statistical tables are clearly set out. There is one typographical error, in the last line on page 17, where "hyperventilation" should read "hypoventilation". A list of references, totalling 90, and from a wide variety of sources, is appended.

[1] "On the Incidence of Anæsthetic Complications and their Relation to Basal Narcosis", by C. J. M. Dawkins, M.A., M.D., B.Chir., D.A., with foreword by J. Blomfield, O.B.E., M.D.; 1936. London: John Murray (for the Middlesex Hospital Press). Demy 8vo, pp. 56 Price: 5s. 3d. net.

VENEREAL DISEASE.

SIX years have elapsed since the appearance of the second edition of David Lees's "Practical Methods in the Diagnosis and Treatment of Venereal Diseases". The third edition, edited and revised by Robert Lees, assistant medical officer for venereal diseases to the Edinburgh Royal Infirmary and Edinburgh Corporation, appears as a tribute to the memory of the author, now deceased.[1]

This excellent volume was written primarily for the student and practitioner, and embodies the methods of treatment practised with success by the author at the Edinburgh Royal Infirmary and elsewhere. In this latest edition recent advances in diagnosis and treatment have been incorporated without appreciably increasing the size of the volume. As formerly, the book is in two parts, the first dealing with syphilis and the second with gonorrhœa. The chapters dealing with syphilis of the nervous and cardio-vascular systems have been rewritten, while that dealing with chancroid, lymphogranulomatosis and other affections less commonly encountered is practically all new material.

The standards of cure of syphilis and gonorrhœa are fully discussed, and stress is laid on the fact, not always fully appreciated, that the disappearance of the signs and symptoms of these diseases does not mean the cure of either.

An interesting feature of the book is the addition of two appendices. One contains a list of the commoner drugs

[1] "Practical Methods in the Diagnosis and Treatment of Venereal Diseases for Medical Practitioners and Students", by D. Lees, D.S.O., M.A., M.B., D.P.H., F.R.C.S., F.R.C.P., F.R.S.E.; Third Edition, edited and revised by R. Lees, M.B., F.R.C.P.; 1937. Edinburgh: E. and S. Livingstone. Crown 8vo, pp. 618, with illustrations, including eight coloured plates. Price: 15s. net.

used in the treatment of syphilis, with their trade names and sources of supply. This should prove of value to those who find some confusion in the multiplicity of proprietary preparations now available. The other contains a list of clinics for the treatment of venereal diseases both in Great Britain and abroad, and is intended for reference in hospital and dispensary practice, so that travellers, seamen and others may be informed where a clinic is available and so secure greater continuity of treatment.

The illustrations and general arrangement are excellent, and the information afforded is lucid and complete. The field is fully covered, and sound principles of diagnosis and practical details of modern treatment will be found throughout. The book may be confidently recommended to those who desire information about the important diseases with which it deals.

RENAL PAIN.

"KIDNEY PAIN: ITS CAUSATION AND TREATMENT", by Dr. J. Leon Jona, of Melbourne, is a carefully compiled record of the observations made from experimental work on the secretory mechanism of the upper urinary tract. The physiology of the passage of urine and the pharmacological action of a number of drugs on the musculature of the kidney, pelvis and ureter are described. Brief mention is made of the clinical application of the experimental findings. Dr. Jona's originality and aptitude for such research are well shown in his book.

In the preface the author states that the chief object has been to bring forward methods to elucidate the cause of obscure kidney pain when the ordinary methods of investigation have failed, and to this he has largely confined his attention.

The importance of pyeloscopy is stressed, and there is no doubt that the routine application of this method in conjunction with retrograde pyelography is of great assistance in the diagnosis of obscure cases of renal pain. No mention is made of intravenous urography, which, when combined with serial photography, should be of considerable assistance in interpreting the physiology of the pelvis and ureter.

This book should prove of value to those interested in urology, but the author presupposes familiarity with cystoscopic and radiographic technique. He is to be congratulated on an excellently compiled little book, the bibliography of which demonstrates the enormous amount of time and thought that has been put into its compilation.

PRACTICAL PHYSIOLOGICAL CHEMISTRY.

IN the laboratory manual of practical physiological chemistry by Wishart, Cuthbertson and Chambers, the class instructions for the course in practical biological chemistry for medical students in the University of Glasgow are given in a compact and convenient form.[2] The little book is on similar lines to that produced by Cramer some years ago for the use of Edinburgh students.

The directions given for each experiment are very brief, but adequate. The book is interpaged to enable the student to add his own comments on and extensions to the directions. Although nothing out of the ordinary is attempted, the course is a very sound one, as far as it goes, and should be well suited, as the authors claim, for teaching "large classes of students where prolonged and involved methods cannot be practised".

Each section is preceded by a short theoretical discussion. Some of these discussions seem out of proportion to the experimental work. Four pages on the classification of the proteins and a folding scheme of the chief derivatives of hæm, for example, seem out of place in a little manual like this.

Sections on the properties of colloids and hydrogen ion concentration are followed by the usual subdivisions on proteins, carbohydrates, lipides, foods and digestion, blood and urine.

DIATHERMY.

THE third edition of "Diathermy", by Elkin P. Cumberbatch, is larger and covers much more ground than the previous edition, and there are several collaborators, as it is no longer practicable for one writer to treat of all the diverse uses of diathermy in medicine and surgery.[1]

Dr. Cumberbatch describes briefly the different kinds of heating by the high frequency current, and gives an interesting history of its use in the treatment of disease. H. J. Taylor deals very thoroughly and clearly with the physics, and there is a full account of the different types of generator of this current.

The technique of applying diathermy to the body and its effects are described and illustrated by numerous experiments. A particularly interesting chapter deals with the treatment of gonococcal infections, and C. H. Robinson gives an account of the use of diathermy in the various types of arthritis and in gynæcology.

H. Wallace Jones deals with cardio-vascular disease, and we are rather surprised to note that he uses general diathermy instead of the auto-condensation couch in treating high blood pressure.

The account of the treatment of *dementia paralytica* by electropyrexia is given by Norman Bell Graham, and should be studied by all those who still infect their patients with malaria.

There is a very good review of the treatment of pneumonia by diathermy, but the great advantages that the inductotherm offers in this field might have been mentioned. The treatment of diseases of the eye, medical and surgical, is given by H. B. Stallard. The ear, nose and throat surgeon also will find that much help is to be had from diathermy.

There are sections dealing with *effleuve* and *étincellage*; short wave treatment also and inductothermy are considered. In the surgical division Dr. Cumberbatch gives the history and describes the types of current used—desiccating, coagulating and cutting. A chapter on the last-named is done by A. H. Burgess, and malignant disease of the upper air passages is dealt with by W. Douglas Harmer.

The late Dan McKenzie defines the usefulness of diathermy in the destruction of tonsils, while Kenneth M. Walker deals similarly with prostatic obstruction.

In a short appendix Dr. Cumberbatch quotes George Crile on the use of diathermy in surgical shock, but does not note the greater convenience and efficiency offered by the inductotherm for that purpose.

This book cannot be too highly recommended as covering the whole field of diathermy, making no extravagant claims, being well written and well illustrated, giving numerous case histories, and being excellently produced.

[1] "Kidney Pain: Its Causation and Treatment", by J. L. Jona, D.Sc., M.D., M.S., F.R.A.C.S., M.C.O.G.; 1937. London: J. and A. Churchill Limited. Large crown 8vo, pp. 190, with 61 illustrations. Price: 7s. 6d. net.

[2] "Practical Physiological Chemistry for Medical Students", by G. M. Wishart, D. P. Cuthbertson and J. W. Chambers; 1936. Glasgow: John Smith and Son (Glasgow) Limited. Crown 8vo, pp. 125. Price: 3s. 6d. net.

[1] "Diathermy, Including Diathermotherapy and Other Forms of Medical and Surgical Electrothermic Treatment", by E. P. Cumberbatch, M.A., B.M., D.M.R.E., F.R.C.P., with nine collaborators; Third Edition; 1937. London: William Heinemann (Medical Books) Limited. Demy 8vo, pp. 592, with illustrations. Price: 21s. net.

The Medical Journal of Australia

SATURDAY, JULY 17, 1937.

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TRUE TO LABEL.

In a small community every practising medical man or woman is a general practitioner, but as the people increase in number certain practitioners devote increasing attention to particular types of ailments, either because they are more interested in them or because they find themselves peculiarly suited to the procedures in diagnosis or treatment that these ailments demand. Eventually there is evolved the type of practice seen in present-day large communities, in which the specialist appears to take the leading part, but in which the general practitioner is the more important unit. This statement is made advisedly, for one can imagine the chaos that would reign in a community that was served entirely by specialists. Those who bow down and worship specialists would do well to remember that specialism has arisen from general practice, that other factors being equal experience in general practice is the best preparation for specialist practice, and that in proportion as specialists lose the ability of looking beyond the confines of their own restricted spheres, so do they lose their effectiveness. In Australia the term specialist has been applied to those whose practice is devoted to internal medicine and to surgery as well as to those whose activities are confined to narrower fields, such as diseases of the eye, of the ear, nose and throat, or of the skin. Perhaps this is just as well, for consultants in the strict sense of the term are practically unknown in this country.

We may set up as an ideal a state of medical practice in which general practitioners send to specialists patients whose illnesses need treatment that only a specialist can give (in this category are included what have been called the minor specialties of ophthalmology, otology, laryngology, dermatology *et cetera*), and in which the advice and help of consultants in internal medicine or in surgery are sought in difficult cases. This state is difficult of attainment and probably never will be attained in a country like Australia, where long distances alone will always be a bar to such an ideal. There is no reason, however, why we should not try to get as close to it as possible. In the way there stand vanity and conceit, hypocrisy and intolerance.

Public hospitals have done a great deal in the differentiation of medical men as practitioners of different specialties. Teaching hospitals recognized as clinical schools have been particularly useful in this way. A senior honorary officer who teaches students in a special department of what is known as a teaching hospital, and who poses as a consultant to whom other practitioners refer difficult cases for elucidation, would be regarded by the most casual observer as a specialist in the subject that he sets up to teach. If the casual observer were to inquire of the attendant at the senior honorary officer's private consulting room, he would probably be told that the said honorary officer was a specialist in the subject taught. Many practitioners of this type, of course, practise their specialty and their specialty alone; others do not. Sir Alan Newton, in his Halford Oration last year, insisted that the cobbler should stick to his last—in other words, that surgeons should be surgeons and not general practitioners. This needs reiteration. It applies not only to surgeons, but also to physicians. Senior

physicians who should know better look with disdain on a brother physician who attends a woman in confinement, but, while posing as consultants, visit patients in their homes in opposition to general practitioners more conveniently situated to the patients than themselves. Of course they always do it "just to oblige the patients". In the practice of medicine less hypocrisy is needed. By all means let us have physicians and surgeons, but let them be true to label. If a surgeon wishes to attend patients with pneumonia just to oblige them, if a consulting physician wishes to visit patients suffering from any and every kind of ailment, let them both be honest and style themselves general practitioners. Other general practitioners will then know where to find their true consultants.

Current Comment.

TRICHINIASIS.

IN Australia trichiniasis (or trichinosis) is of little more than academic interest. A. J. Bearup, in THE MEDICAL JOURNAL OF AUSTRALIA of April 3, 1937, gives an account of a search for *Trichinella spiralis* in cadavers in Australia. He concludes that no instance of this nematode's having originated in Australia has been demonstrated. However, in view of the importation of pigs, it is impossible to be certain that this immunity will endure. Accordingly it is essential that some certain method of diagnosis should be established. Trichinosis is notoriously difficult to diagnose, and errors of diagnosis have often occurred. Eosinophilia generally is present, but may be absent. Typhoid fever and malaria are the diseases most often confused with trichinosis. One case has been recorded in which acute glomerular nephritis was diagnosed, and another simulated thrombophlebitic septicæmia. Patients with cerebral signs and symptoms may show the nematode in the spinal fluid, but this is inconstant. Several observers have recorded instances in which symptoms were referable to the central nervous system, and in one instance, in which larvæ were discovered in the blood as well as in muscle sections, toxic psychosis and encephalitis were manifested. At the autopsy trichinellæ were seen in the myocardium, pancreas, kidney and intestinal mucosa. In another instance generalized peritonitis was found *post mortem*. In view of the difficulties of diagnosis, a reliable test has been searched for. Fülleborn's skin reaction has been strongly positive in some cases.

A. A. Hall gives an account of an outbreak of trinchiniasis in Ohio, and refers particularly to the use of the Bachman intradermal skin test in diagnosis.[1] From August, 1928, to March, 1936, ninety-six cases were reported in Ohio, with eight deaths. Hall states that comparatively few reports regarding the use of the Bachman test are to be found in the literature, although it was first described in 1928. Hall thought that the test was valuable in the diagnosis of trichiniasis and that it should supplant the biopsy of muscle. Bachman described the preparation of his antigen thus:

> The larvæ are separated by artificial digestion and thoroughly washed free from host proteins, dried at 47° C. in a vacuum over sulphuric acid and thoroughly pulverized. Test antigen is prepared from a 1 per cent suspension of the dried pulverized trichinella powder, extracted at room temperature for a day or longer in Coca's solution, then centrifuged at high speed for 15 minutes and the supernatant fluid used as the test antigen.

Hall reports six cases constituting an epidemic group, and also two which occurred at the same time, but in which the patients were infected from a different source. The Bachman skin test was also applied to a series of thirteen control patients who were admitted to hospital for other diseases and who gave no indication of trichiniasis. All of these gave entirely negative responses, except one, whose response was as strongly positive as any of the definitely infected individuals. This patient gave a history of having eaten raw sausage sandwiches for many years and had suffered from vague muscular or arthritic pains. She did not remember ever having had œdema. She had an eosinophilia of 4%. She had been admitted to hospital for chronic cardiac disease, and Hall, with some justification, regards her condition as a case of trichiniasis revealed by the Bachman test. Hall believes the test to be very satisfactory, and he regards it as equal in value to the average diagnostic skin test in other fields. He obtained positive reactions in every one of his cases. All patients had eosinophilia ranging from 31% to 85%, except one, whose eosinophilia was only 5% on his admission to hospital, but rose to 31% ten days later. The accuracy of the skin test depends upon securing a suitable preparation of powdered larvæ and properly preparing the antigen solution from the powder. At present there is no adequate supply of the material available. New antigen must be prepared frequently. When kept on ice, it is held to retain its potency for about six months, but it deteriorates much more rapidly when not so preserved. Hall found one preparation inert after two months. Other observers have also obtained satisfactory results. O. R. McCoy and his colleagues applied the test to 88 persons who had been infested within the previous eight years. Of 36 of these who had been ill two to six weeks, 31 gave a positive result to the test. Of 39 tested from three to twenty-two months after infestation, 31 gave positive reactions. Of 13 who were ill three and a half to seven and a half years prior to the making of the test, eight gave positive reactions. McCoy applied the test to 539

[1] *Annals of Internal Medicine*, April, 1937.

controls, of whom only seven gave positive reactions. It would seem from McCoy's results that the test approaches specificity in the early stages, but that sensitivity diminishes with the lapse of time.

Hall did not employ Bachman's precipitin test. He states that this test does not usually give a positive result until three or four weeks after infection. However, he considers that the test would be of use in detecting border-line cases which are of greater duration than this. Bachman himself claimed that the precipitin test was specific in 90% of cases.

Writers have described a "trichiniasis facies" in sufferers from that disease. This consists essentially of œdema of the eyelids with œdema and hyperæmia of the conjunctiva, especially over the outer part of the eyeball. Hall does not specifically mention this facies, but in his cases œdema of the eyelids, especially of the lower lids, or even generalized œdema was present. Bilateral conjunctivitis, sometimes with photophobia and redness round the orbit was also noted. Other manifestations were cramp-like pains in the legs or generalized muscular pains or profound muscular weakness. Dysphagia, laryngitis, vomiting or diarrhœa were also observed, as well as rigidity of the abdomen with tympanites and tenderness over the liver and spleen. Other observers have recorded an abdominal rash resembling that of paratyphoid fever. Hall does not mention such an occurrence.

IRON-DEFICIENCY ANÆMIA IN INFANCY.

THERE is perhaps some impression amongst the readers of medical literature that recent researches in hæmatology have tended to complicate matters and that anæmia is by no means a simple study. This is, in essence, perfectly true; yet it is also true that the most important and common types of anæmia are of a type easily comprehended and frequently also easily treated. Obviously the chief point is their recognition. It is so evident a thing that young children are frequently "run down" after infections, for example, that this commonplace has not received due attention, and it is safe to assert that few practitioners have troubled to ascertain the blood state of very young children unless they are keen pædiatricians. H. W. Fullerton has recently published an article on the iron-deficiency anæmia of late infancy; and, coming from the Department of Medicine in Aberdeen, known to be a centre of great activity in the study of blood diseases, this work is significant.[1]

The extensive survey of the blood state of the poor classes of Aberdeen carried out by workers in this department enabled Fullerton to collect the results of hæmoglobin estimations of the blood of 789 infants of all ages below two years. Part of these results have already been published, but the full records now confirm the impression previously gained that infants from nine months onwards frequently suffer from anæmia under such conditions as prevail amongst a poor population in a large city. Naturally we next inquire what is the cause. Fullerton gives the causes as (*a*) the type of milk feeding, (*b*) the effect of maternal iron deficiency, (*c*) the birth weight, and (*d*) the effect of infections. As regards the first category exact figures are difficult to obtain, but the author thinks there is good evidence for believing that the breast-fed baby retains at the end of his first nine months a quantity of iron that is definitely higher than that retained by artificially fed babies. Maternal iron deficiency has been thought by many observers to predispose infants to anæmia towards the end of the first year, though it does not influence the hæmoglobin level of the infant at birth. This question is thoroughly discussed in this paper, but the conclusion reached is that a maternal iron deficiency is not a very significant factor in causing infantile anæmia. Needless to say, this would not in any case affect the necessity for efficient treatment of anæmia occurring in the expectant or nursing mother. Birth weight is a matter of greater importance. Figures clearly show that the lower the birth weight, the lower the hæmoglobin level in late infancy, owing to the smaller iron content of circulating hæmoglobin in the smaller baby, who tends to grow more rapidly in proportion to his birth weight. Thus the iron content of the blood depends upon blood volume, which in turn depends upon body weight; so that we have in the baby's birth weight a rough measure of his available iron. This generalization is of great value, as the practitioner may thereby be on his guard in the case of small or premature babies. The last point, the effect of infections, is also most important. Some simple but striking charts in this paper illustrate this well known but very vital factor. Just how infections act in reducing the hæmoglobin level is not clear, but the mechanism would seem to be in all probability an inhibition of blood formation. Here one is reminded of the old wives' fondness for "Parrish's food" for little children after illness, an empirical form of treatment which is not far removed from the giving of a solution of ferrous sulphate in 50% glucose common in Aberdeen. An important point emerging from the inquiry into the effect of infections is that the inhibitory influence on blood formation is apparently felt for some considerable time after all clinical signs of the infection have disappeared. The estimation of hæmoglobin is a very simple procedure. To carry out this test accurately and to determine correct standards is by no means so easy, but an approximation is simple to arrive at, so that there would seem to be little reason why anæmia in late infancy should be overlooked. Easily caused and easily cured, it should be looked for even in children who are not obviously ill, and its recognition should lead to the attainment of a higher standard of health in the later months of infancy, when the diet often tends to be low in iron, particularly among the less fortunate members of the community.

[1] *Archives of the Diseases of Children*, April, 1937.

Abstracts from Current Medical Literature.

RADIOLOGY.

Multiple Tumours Within the Spinal Canal.

JOHN D. CAMP (*American Journal of Roentgenology and Radium Therapy*, December, 1936) states that in a recent series of 72 cases in which tumours affecting the spinal cord were localized by means of myelography, in 4% of the cases the lesions were multiple and were so diagnosed by X ray examination before operation. When two cubic centimetres of lipiodol are distributed along the subarachnoid space, pseudo-arrests which simulate a partially obstructing lesion are not uncommon. At least five cubic centimetres of oil are indicated in order to fill the subarachnoid space completely at any desired level and to reveal the tumour by virtue of its filling defect before obstruction has occurred. If the radiological examination is limited to the preparation of skiagrams alone, and obstruction only is looked for, many partially obstructing or non-obstructing lesions, some of which may be multiple, may be overlooked. A thorough study must be made of the excursion of the oil, with the patient in the prone, supine, oblique and lateral positions on a tilting examination table. By careful study of the shape of the shadow cast by the oil and of the nature of the defect observed, it usually is easy to determine whether a tumour is extradural, subdural or intramedullary, and frequently the position of the tumour as related to the anterior, posterior and lateral aspects of the cord can be elicited. These points, of course, may be of great importance in predicting the nature of the tumour, particularly in the case of a protruded intervertebral disk.

Osteochondrosis and Osteochohdritis Ischio-Pubica.

A. A. ZEITLIN (*Radiology*, December, 1936) considers that the disease that is at present described under the name of *osteochondritis ischio-pubica*, according to clinical and X ray findings should be divided in two groups. The first is nearer to the picture seen in osteochondropathy of juvenile age, and can be designated as *osteochondrosis ischio-pubica*; the second has at its base a chronic inflammatory process and retains the designation of *osteochondritis ischio-pubica*. The prognosis in osteochondrosis is quite favourable, and no special methods of treatment are required. In osteochondritis the prognosis in general is also favourable, but the bone lesions are much greater and the deformation in the symphysis can possibly play a certain rôle in the future for girls during childbirth. In *osteochondrosis ischio-pubica* there appears in the region of the synchondrosis an oval or round focus of rarefaction, sometimes of crumb-like structure, with distinct contours. The focus, which rarely spreads over the entire width of the bone, leaves strips of normal tissue on the medial and lateral sides, sometimes even condensed on the side of the *angulus pubis*. Very characteristic is the projection of the bony cartilaginous mass in the form of a triangular promontorium in the lumen of the *foramen obturatorium*. Such a promontorium is sometimes observed as a variant in a case of normal ossification, but it does not reach such large proportions as in *osteochondritis ischio-pubica*. The edges of the decalcified zone on the side of the *os pubis* and *os ischii* remain well defined and become condensed in some cases and appear sclerotic in character. The adjoining bone is normal. The focus remains in such a condition for from a few weeks to several months; then begins a gradual process of restitution, the bone taking on a normal appearance, and only the promontorium into the lumen of the *foramen obturator*, retarded in its restitution, remaining of the former process. In *osteochondritis ischio-pubica* the pathological process is more clearly revealed with regard to the intensity of the changes and their development. The process of decalcination and dystrophy is not limited to the synchondrosis, but is transferred to the adjoining part of the *os pubis* and *os ischii*, forming one continuous focus or appearing as a number of separate spots. There are no reactive manifestations or they are very slight. The process is of longer duration, continuing from several months to years. In far-advanced cases the deformation of the bone is quite considerable, and this stands as a contradiction to the complete absence of clinical symptoms.

Traumatic Separation of the Lower Epiphysis of the Radius.

C. L. GILLIES (*American Journal of Roentgenology*, February, 1937) states that traumatic separation of the lower epiphysis of the radius with an incidence of 22% of the bony injuries in the region of the wrist before the closure of the epiphysis is important, first, because of its frequent occurrence and, second, because of the possibility of resulting arrest of growth. In the author's series this occurred in 14% of the separations. Lack of full reapposition of the epiphysis after displacement seems to play a part in the development of shortening in some instances. Excepting those rare instances of longitudinal fracture of the epiphysis in which, from the very nature of the injury, arrest of growth is prone to occur, there is no way of predicting either from a clinical or radiological study whether or not deformity will develop or, if a deformity is present, whether it will disappear in time. In all cases in which the patient is seen soon after displacement has occurred, early reduction under anæsthesia should be attempted. In those cases in which the patient is seen late and in which vicious union has occurred, reduction should not be attempted, for, apart from being likely to be unsuccessful, attempts at reduction may further injure the epiphyseal cartilage so that it cannot overcome a deformity which it might otherwise overcome. Whether resulting deformity is present or not, the prognosis for a painless, useful wrist is excellent.

X Ray Evidence of Metastatic Malignant Disease in Bone.

HENRY SNURE AND GEORGE D. MANER (*Radiology*, February, 1937) state that it is surprising how frequently metastatic malignant disease is present in the vertebræ without clinical signs and without evidence on skiagrams that have excellent detail and quality. They removed a V-shaped section of bone from the vertebral bodies of all patients dying of malignant disease when autopsy was permitted. To avoid distortion due to the spine-film distance or to superimposed shadows, the spinal column was sawn lengthwise in the sagittal plane and one half was placed directly on the film holder; even under these conditions the larger areas of destruction were not demonstrable on the film. To determine the size of an area of metastasis that could be visualized on the X ray film, portion of the spongiosa of the vertebral bodies was removed and the cavity was filled with muscle scraps, water or paraffin. Spongiosa occupying an area of 2·0 by 2·5 by 2·75 centimetres and 1·0 centimetre deep can be removed from a vertebral body, and skiagrams made after this space has been filled with paraffin give practically no evidence of change in the spongiosa. It would be impossible to visualize on skiagrams an osteolytic tumour of the above size in the vertebræ of a living person; fine detail of the spongiosa would be partially obliterated by the film-vertebral distance and by the superimposed soft tissue shadows. Apparently the cortical bone accounts for most of the detail of the osseous structures depicted on the film, while the spongiosa accounts for the general density. When areas of cortical bone 1·0 centimetre square are removed from the lateral surface of the vertebral body and the excavated area is filled with paraffin, their location can be seen on the skiagrams. Holes, made by a trephine 1·0 centimetre in diameter and extending almost across the vertebral body after being filled with paraffin, are not visible unless the central ray passes through the long axis of the hole; and they are not visible then if the cortex is replaced. Similar findings can be demonstrated in other bones of the body. Films made of the tibia, with spongiosa of the marrow cavity (2·0 centimetres in diameter) removed and filled with paraffin, show no

change when compared with films of the tibia before removing spongiosa, the soft tissues (such as muscle) remaining in place in both instances. Likewise, an *os calcis* can be sawn lengthwise into halves, the spongiosa can be curetted out, and a space from 1·5 to 2·0 centimetres in diameter can be filled with paraffin, and no evidence of change will be apparent on the subsequent film. Ribs sawn lengthwise and marrow curetted out show no deviation on the film from the normal. With the ribs no paraffin was used and it was not necessary that the halves of the ribs should fit accurately upon each other. The dense crescentic shadow usually found on films that depict ruptured *nucleus pulposus* is probably due to a slow rebuilding of new bone at the rupture site rather than to a piling up of the small fragments of the trabeculæ of the spongiosa displaced by the cartilage. It seems reasonable to assume that in a recent rupture of the *nucleus pulposus* no X ray evidence of rupture can be demonstrated on the film.

X Ray Diagnosis of Lipomata.

FREDERIC E. TEMPLETON (*American Journal of Roentgenology*, February, 1937) states that since the X ray absorption coefficient of fat is less than that of water, it is inevitable that fat should absorb less radiation than the other non-skeletal tissues, because the latter are made up largely of water. Lipomata containing sufficient fat cast sharply demarcated oval or round shadows less dense than those cast by surrounding or neighbouring soft tissues and body fluids. This appearance is of great help in differentiating these tumours from other types of soft tissue neoplasms and from collections of fluid, such as cold abscesses.

PHYSICAL THERAPY.

Treatment of Prostatic Carcinoma.

B. S. BARRINGER (*American Journal of Roentgenology and Radium Therapy*, January, 1937) offers some suggestions as to (i) the points of origin of prostatic carcinoma, (ii) diagnosis by aspiration biopsy, (iii) pathological anatomy influencing radiation. He deduces that carcinoma, in the great majority of cases, originates in the posterior lobe, and in most cases there is one focus of origin. The posterior lobe is the most convenient place for aspiration biopsy. The proper use of aspiration biopsy in early suspected cases of prostatic carcinoma is one of the important factors in solving the question of control. Considering the great difficulties of operative removal of the carcinomatous prostate and the comparatively small percentage of cures, it has been necessary to turn to some other method, and that has been irradiation. The best method of deep X ray therapy, in which 300 *r* are given daily to four or five portals, each portal receiving 1,500 to 1,800 *r*, is not sufficient to control the disease. External irradiation must be supplemented by some form of irradiation within the prostate itself. The author advises suprapubic implantation of radon seeds into the prostate.

Radium Treatment of Fibroid Disease of the Uterus.

R. GAUDUCHEAU (*French Journal of Radiology*, January, 1937) states that in the past, treatment of bleeding and moderately large fibroids, of *fibrosis uteri* and of hæmorrhagic metritis has been carried out by X ray applications. The greater number of these patients are referred by the surgeon, who naturally hesitates to operate on a patient already exsanguinated. The urgent necessity for rapid and efficacious treatment and the difficulties of transport of large X ray apparatus are the reasons why radium has lately been preferred to X radiation. In the description of his technique the author stresses the fact that of late years he has increased the amount of radium placed within the uterine cavity and has considerably lessened that within the vagina, by this means avoiding secondary atresia. The usual dosage administered is twenty millicuries destroyed within the uterus and thirteen in the vagina. The tubes are removed once or twice during the course of application to permit of vaginal douching and the changing of tampons. In a series of twenty-nine cases one patient died from a pulmonary embolus. Of the remaining twenty-eight, two (in 1922 and 1923) received too small a dose of radon and the bleeding returned after six months, although much less in quantity. The remaining patients passed through the normal cycle of an artificial menopause.

Radium Treatment of Carcinoma of the Rectum.

H. H. BOWING AND R. E. FRICKE, of the Mayo Clinic (*Journal of Radiology*, May, 1937), state that careful study of each individual patient is necessary in carcinoma of the rectum before a plan of treatment can be formulated. In well-selected cases, in which lesions are operable, pre-operative radium treatment offers a great deal. In inoperable lesions radium therapy secures palliation, increased length of life and comfort to many patients whose condition otherwise is hopeless. In the authors' series of cases, 132 in number, 60% of patients had carcinomata that were inoperable. Types of radium treatment fall into two general categories. The first type is radical aggressive treatment intended to effect a possible cure; the second is limited palliative treatment. The techniques used consist in implantation of seeds or needles and contact treatment by the use of radium or radon tubes strapped together to form a plaque; palliative treatment consists in external irradiation by radium or deep X rays, central irradiation with a radium-containing tube in the lumen and vaginal irradiation in women. Colostomy is optional except when obstruction is present, but is very valuable. Treatment is necessarily tedious and reward of good results varies directly with the care and judgement exercised.

Radiation Treatment of Inoperable Intraabdominal Malignant Disease.

E. A. MERRITT (*American Journal of Roentgenology and Radium Therapy*, September, 1936) gives a preliminary report on a series of thirteen patients with inoperable intraabdominal cancer treated since January, 1934. Three are living and apparently well and free of all evidence of disease, X ray examinations having been made within a month prior to the writing of the article. One is living, but shows radiological evidence of disease. Nine are dead of the disease. The patients have been taken for treatment without regard to their condition or to the extent of involvement of the stomach, and most of those who succumbed were in a dying condition when treated. This statement also applies to one of the three who is living and well today. The author has learned, much to his surprise, that patients suffering from malignant disease of the stomach tolerate treatments very well. Treatments were given daily after the modified Coutard technique. In three of the living patients the tumour doses were respectively 2,100 *r*, 2,200 *r*, and 2,500 *r*, delivered by means of about twenty-four treatments.

The Radiological Treatment of Malignant Tumours of the Oral Cavity and Pharynx.

ELIS G. E. BERVEN (*Acta Radiologica*, February, 1937), discussing malignant tumours of the oral cavity and pharynx, describes the method of treatment employed and the results obtained in the Radiumhemmet during the years 1916 to 1930. In the course of that long period the methods have gradually developed into the technique now in use, the particulars of which the author sets forth and discusses in detail. As a rule the beginning is made with teleradium treatment from several fields of entry. In most cases the dose applied produces the mucosal and cutaneous reactions described by Coutard (epitheliitis and epidermitis). When the period of reaction is past, usually after six weeks to two months, any possibly remaining remnants of the primary tumour are dealt with locally, by surgical excision, electrocoagulation or interstitial implantation. If any lymph gland metastases persist after the reaction, they are dissected *en bloc*. Of 457 patients with oral cavity tumours treated, 114 (25%) lived, free from symptoms, for five years or more. Of 39 patients with carcinoma or lymphepithelioma of the tonsils treated, 16 (41%) lived free from symptoms for five years or more. Of 49 patients with sarcoma of the tonsils treated, 17 (35%) lived, free from symptoms, for five years or more.

British Medical Association News.

SCIENTIFIC.

A MEETING of the South Australian Branch of the British Medical ASSOCIATION was held at the Anatomy Theatre, University of Adelaide, on April 29, 1937, DR. A. F. STOKES, the President, in the chair.

Peptic Ulcer.

DR. F. H. BEARE read a paper entitled "Peptic Ulcer" (see page 86).

DR. IAN HAMILTON read a paper entitled "Some Remarks on the Treatment of Peptic Ulcer" (see page 94).

DR. A. R. SOUTHWOOD regretted that the lateness of the hour prevented full discussion of the two very excellent papers. Whenever the subject of peptic ulcer came up for review, great interest was always shown in discussing the relative merits of medical and surgical methods of treatment. The treatment outlined by Dr. Beare was rigorous, yet any in that room unfortunate enough to be afflicted with a peptic ulcer would far prefer to follow such a *régime* at the outset of trouble, rather than to risk the later development of complicating features likely to require some drastic method of surgical attack, such as those referred to by Mr. Ian Hamilton. It would be agreed that medical management was suitable for patients with uncomplicated peptic ulcer, but that operative treatment might be necessary in the very chronic or complicated cases.

PROFESSOR J. B. CLELAND showed tables giving the findings of gastric ulcers including scars, and duodenal ulcers including scars, in 3,000 autopsies at the Adelaide Hospital, commencing in the year 1920, together with the ages and sexes of the patients. These are shown in Tables I and II. He pointed out that in all autopsies the stomach and duodenum had been regularly examined, though perhaps in the first thousand this had not been so thoroughly done as in the later autopsies. In most cases in which scars only were present it had not been possible to suggest how old they were, though one of the males under "Duodenal Ulcer", who was in his teens, showed a scar. The ages shown did not indicate the exact ages at which ulceration commenced, or even was present, owing to the inclusion of scars of old ulcers and the fact that an ulcer might be present for many years before death. It would be noted, however, that some of the duodenal ulcers were in unusually young people and that the age distribution was somewhat younger than that of gastric ulcer. It would be also noted that in Professor Cleland's experience in South Australia gastric ulcers or their scars were definitely commoner than duodenal ulcers and their scars. *Post mortem* examinations were done on approximately two males to one female. The figures both for gastric ulcer and duodenal ulcer showed, therefore, a very decided preponderance in men over women.

DR. H. A. MCCOY said that of the difficulties which arose in the diagnosis of peptic ulcer, he wished to discuss two problems which had exercised his mind considerably. Perhaps Dr. Beare would be prepared to answer these questions. (i) When was a duodenal ulcer healed? (ii) Was it possible to differentiate the benign from the malignant ulcers seen on the lesser curvature or the posterior surface of the stomach?

Dr. McCoy believed that it was very difficult to answer the first question from the radiological point of view. The evidence of active ulcer was reliable when a definite niche was found associated with characteristic deformity of the cap, surging in the duodenum and tenderness on pressure. But what of the cases in which, after a course of medical treatment, some of these signs were still present? If the symptoms had disappeared, it would be reasonable to suppose that the persistent deformity was due to simple scarring. If a niche persisted and if symptoms had subsided, it might be interpreted as a depression of the scar. But really satisfactory evidence of healing was achieved only when the cap, originally deformed and with a niche, was visualized as a normal filling. Such evidence in Dr. McCoy's experience was rare.

It would seem, therefore, that the duodenal ulcer was, like the poor relation, always with them. The intermittent exacerbation of symptoms indicated, of course, the tendency of these ulcers to recur, and they had learned from experience that long-continued and rigid treatment was the only means of checking these exacerbations. It would appear that once gross ulceration of the mucosa had occurred in the duodenum, the possibility of solid healing which would allow of a normal mode of life, was remote. Dr. McCoy admitted that his views might be distorted by reason of the selection of patients who were sent for reinvestigation, and if this was so, he would be grateful for enlightenment.

TABLE I.

Type of Lesion.	First Thousand.	Second Thousand.	Third Thousand.	Total.
Gastric ulcers, including scars	18	23	26	67 (51 males, 16 females)
Duodenal ulcers, including scars	12	15	20	47 (39 males, 8 females)

TABLE II.
Ages.

Lesion.	10's.	20's.	30's.	40's.	50's.	60's.	70's.	80's.	Age Unknown.	
Gastric ulcer:										
Males	—	2	4	7	12	16	7	2	1	51
Females	—	—	1	3	6	2	2	1	1	16
Total										67
Duodenal ulcer:										
Males	3	1	4	11	6	8	5	1	—	39
Females	—	—	3	1	—	3	1	—	—	8
Total										47

Malignant ulcers might be, he believed, differentiated with reasonable accuracy, and as early selection of treatment was important, the value of the differentiating signs should be recognized. The typical penetrating ulcer, associated with converging rugæ and uninterrupted peristalsis, would invariably fit in with the clinical and test meal findings of simple peptic ulcer. The ulcer, usually on the posterior surface, associated with a niche (possibly only a small one), with the rugæ disposed around the crater in the form of a halo and with interruption of peristalsis over this segment, was the classical picture of a malignant ulcer. In this type of carcinoma, which tended to ulcerate at an early stage, Dr. McCoy believed that the condition might be diagnosed at a stage when it was operable.

The healing of the gastric ulcer might occasionally be studied radiologically, and it was his impression that complete healing might take place without any considerable deformity of the gastric rugæ, and certainly without hourglass deformity, which was, Dr. McCoy believed, a rarity.

The need for correlation of test meal and X ray findings of peptic ulcer was, he suggested, very important. In order to extract the maximum value from radiological investigation, a satisfactory visualization of gastric rugæ was essential, and this might be achieved best when the stomach was empty. It would seem, therefore, that the ideal time for X ray investigation was immediately after the completion of the test meal, when a small amount of barium was administered for the purpose of demonstrating the rugæ. This sequence was followed by several Continental authorities, who claimed distinct advantages for their method.

Dr. McCoy said that Dr. Ray Hone and he had endeavoured recently to collaborate in this respect; and, though their experience of the method was so far small, he felt that the method had much to commend it, especially, of course, in cases in which there was slow evacuation of the gastric contents.

DR. ALAN LENDON reemphasized the necessity for collaboration between the surgeon and the physician and the radiologist. He drew attention to the fact that occlusion of the duodenum, performed at the same time as the gastro-enterostomy, forced the latter to function.

DR. H. R. POMROY said that peptic ulcer was primarily a disease for medical treatment and that operation should not be performed except in the presence of complications or in special circumstances. He advised adequate medical treatment for at least two years, and said that the prognosis depended on the length of time that symptoms had been present. He stressed the point that ordinary X ray films were not always satisfactory, but that screening was essential in order to obtain the maximum amount of information.

DR. E. MCLAUGHLIN stressed the necessity for obtaining records of the secretory activity of the stomach as an aid to correct diagnosis, pointing out that conditions associated with achlorhydria not infrequently induced a pain difficult to distinguish from that experienced in peptic ulcer. Dr. McLaughlin criticized the customary "alkaline" treatment and insisted upon the necessity for an adequate vitamin ration.

In reply, Dr. Frank Beare said how pleased he was to have been associated with Dr. Ian Hamilton in the preparation of the evening's paper. His only complaint was that so much had had to be left unsaid by both Dr. Hamilton and himself.

In reply to Dr. Southwood, he said that he agreed that the medical treatment would be even more successful if it were carried out for longer periods. It was absurd that many patients medically treated should be discharged from hospital at the end of four weeks. No two ulcers behaved in exactly the same way when treated. He was somewhat startled when he first saw Professor Cleland's figures for the occurrence of peptic ulcer, showing as they did that gastric ulcer was more commonly met with at *post mortem* examination than the duodenal variety. Professor Cleland had included scars as well as definite ulcers, but that did not explain the discrepancy, for presumably the scars had at some time or other been ulcers. Most series of cases showed that duodenal ulcer was more frequently seen at autopsy than gastric ulcer.

Dr. McCoy had raised the question of the healing of ulcers and how this could be judged clinically. In the first place, ulcers definitely did heal, as was evidenced by Professor Cleland's *post mortem* findings. It was difficult clinically to know when an ulcer was healed. The cessation of symptoms was an unreliable guide, for in an uncomplicated case this should come about during the first week of treatment. The repeated absence of occult blood from the fæces carefully examined suggested that healing was taking place or had actually come about. The X ray findings, such as definite decrease in the size of the ulcer crater, lack of tenderness when the ulcer area was pressed during a screening, and absence of the indirect evidence of active ulcer suggested healing. About the last Dr. McCoy had told the meeting previously and there was no need to repeat what had been dealt with so ably by him. Nevertheless, the subject was a difficult one, and Dr. Beare suggested that six weeks was a minimum period for healing to take place.

Dr. McCoy had also asked how a simple ulcer could be distinguished from a malignant ulcer. Again, this was a difficult question to answer, and in itself would prove an interesting subject for a whole evening's discussion. Dr. Beare thought that there were two distinct problems bound up into this question. One had to consider the question of an ulcer malignant from the outset and, on the other hand, a simple ulcer that had taken on neoplastic features. In the former case, as Osler had so frequently pointed out, many such ulcers were associated with a relatively short history of digestive upset. Not so the latter, for the usual history was one of long-standing "indigestion", in other words, the usual ulcer history.

The test meal was of great value, but not conclusive, for with some gastric ulcers there was a low hydrochloric acid content; but, other things being equal, a normal or high hydrochloric acid content was in favour of simple ulcer. The X ray findings were helpful, for the presence of an area around the ulcer over which the peristaltic wave was held up, no doubt owing to infiltration round the ulcer, was in favour of a neoplastic ulcer. The size of the ulcer was suggestive, anything apparently larger than one inch in extent was suggestive of a neoplastic change. Trial by treatment was useful, but it should be remembered that the medical treatment of simple ulcer relieved symptoms of carcinoma of the stomach in quite an appreciable number of cases, for some weeks at any rate. The final test, however, was the histological examination of the results of gastrectomy, and in case of doubt there was every justification for recommending such an operation. Partial gastrectomy was definitely indicated in many cases of gastric ulcer, as Dr. Hamilton had pointed out, and it was better to be safe and not to "sit on" an early carcinoma. Of course, it was unnecessary to point out that primary carcinoma of the duodenum was a pathological curiosity and so did not need consideration in this discussion. Dr. Beare agreed with Dr. McLaughlin, who had said that many patients were treated as though they suffered from ulcer when there was little evidence to show that this condition was present. He thought that he had made it clear that much of the hydrochloric acid was fixed by the food after a meal. He agreed that bicarbonate of soda was not altogether a suitable alkali, for it unfortunately stimulated acid production. As regards the action of bismuth in covering the ulcer with a protective layer, he did not think this was very likely to occur, for screening after the bismuth powder was taken therapeutically rarely showed this substance adhering to the ulcer.

There was a definite risk of mild scorbutic symptoms developing when a strict diet had been adhered to for any length of time. This was best dealt with by using fruit juice, a feature of some *régimes*.

Dr. Beare was very cheered to hear Dr. Pomroy state that peptic ulcer was primarily a disease for medical treatment. This statement coming from a surgeon was most refreshing. He quite agreed with Dr. Pomroy when he said that screening was absolutely necessary for the

accurate diagnosis of peptic ulcer. Unfortunately in the practice of some hospitals this was a counsel of perfection. On the other hand, Dr. Beare knew that many patients were submitted to unnecessary X ray examination and more or less as a forlorn hope. This was not altogether the fault of the hospital staff.

A MEETING of the Victorian Branch of the British Medical Association was held at the Royal Melbourne Hospital, Melbourne, on Wednesday, May 19, 1937. The meeting took the form of a number of clinical demonstrations by members of the honorary staff. Part of this report appeared in the issue of July 10, 1937.

Treatment of Erysipelas.

DR. G. R. A. SYME showed a series of charts depicting the results of treatment of erysipelas by sulphonamide. The charts showed a more rapid subsidence and an absence of complications when compared with a control series.

Paralytic Ileus.

Dr. Syme also demonstrated a model of the treatment adopted for paralytic ileus, consisting of an apparatus for continuous gastric suction, continuous intravenous drip of saline and glucose solution, the four-hourly administration of morphine (one-sixth of a grain), and the application of heat to the abdomen. The results of the treatment for a short series had been satisfactory.

Polyposis of the Colon.

Dr. Syme also demonstrated a specimen of multiple polyposis of the colon, showing the presence of two distinct carcinomatous growths and several innocent papillomata.

Hysteria.

DR. N. A. ALBISTON demonstrated a case of war neurosis. The patient exhibited coarse functional tremor of the head and hands and a high-stepping gait, which had been present since shell-shock in 1917. The condition prevented the patient from following any permanent occupation and had necessitated the constant use of a walking-stick. The history antedating the shell burst which precipitated the symptoms, was a typical one of intolerable mental distress through conflicting fear and duty impulses. The patient proved to be a somnambule under hypnosis and was cured in a few minutes after a psychological analysis had revealed the mechanism of his symptoms. The importance of analysis and careful history-taking before the employment of suggestive therapy was emphasized. Hypnosis itself gave no guarantee of permanency in the absence of attention to all factors present in the genesis of the case. Dr. Albiston demonstrated hypnotic control over the patient.

Eventration of the Diaphragm.

DR. IVAN MAXWELL presented a female patient, fifty-four years of age, who had complained of anorexia, dyspnœa on exertion and a choking sensation in the throat, accompanied by slight retrosternal pain. The symptoms had been present for four months and the patient had lost twenty-eight pounds in weight. In adolescence she had had pleurisy of the left lung, and in 1928 her left breast was removed for carcinoma. Physical examination revealed the absence of breath sounds at the base of the left lung, with dulness on percussion. The heart showed slight enlargement, with two fingers' breadth of right cardiac dulness; the diastolic blood pressure was 115 and the systolic pressure 180 millimetres of mercury. On radiographic examination it was reported that there was moderate diaphragmatic excursion on the right side and poor excursion on the left. Eventration of the left side of the diaphragm was the interpretation, with widening of the upper mediastinal shadow, due to the fact that the aorta was widened and uncoiled; the lower mediastinum was pushed to the right by eventration of the left dome of the diaphragm; there was no evidence of enlargement of the mediastinal glands due to secondary glandular involvement from the original carcinoma of the breast.

Paget's Disease.

Another patient shown by Dr. Maxwell was a woman, fifty-nine years of age, who had complained of pain in the right leg, extending from the hip to the ankle. The pain was pronounced during rest and diminished with exercise; it had been present for approximately twelve months. Examination revealed considerable outward curvature of the right leg and to a lesser degree of the left leg; there was a shortening of one inch in the right leg, and there was limitation of movement at the right hip joint, especially on adduction. Apart from the osseous system no abnormality was found. Dr. Maxwell said that radiographic examination revealed extensive evidence of Paget's disease, most pronounced in the bones of the pelvis and the femora; there was loss of joint space with early lipping in both hip joints, preponderating on the right side.

Pernicious Anæmia.

Dr. Maxwell showed a male patient, seventy-five years of age, who had first come under his notice in August, 1935, when he complained of pain in the precordium and legs, with sickness and dyspnœa on exertion, of five weeks' duration. Vomiting had occasionally occurred and the patient had lost twenty-eight pounds in weight in twelve months. The patient had a history of prostatectomy in 1928, and had been treated for pernicious anæmia two years afterwards, for five months, but had had no treatment since then. It was reported on examination that the heart was slightly enlarged and that a soft systolic bruit was present at the mitral area. The systolic blood pressure was 150 and the diastolic pressure 75 millimetres of mercury. There were crepitations at both bases of the lungs; the liver was enlarged and tender; no free hydrochloric acid was found in the stomach, even after the injection of histamine. A blood count revealed 470,000 red cells per cubic millimetre, 3,200 white cells per cubic millimetre, and a hæmoglobin value of 30%; the reticulocytes were less than 1%, and the colour index was estimated at 1·07. Thirty-two cubic centimetres of "Campolon" were injected between August 14, 1935, and August 30, 1935, by which time the red blood cell count had risen to 3,360,000 cells per cubic millimetre; the colour index was 0·81, and the hæmoglobin value was estimated at 54%. A subsequent injection of ten cubic centimetres of "Campolon" was sufficient to restore the blood function almost to normal, and this was maintained by monthly injections of five cubic centimetres of "Campolon". On March 6, 1936, the red blood cell count had risen as high as 5,103,000 cells per cubic millimetre, and the hæmoglobin value was estimated at 72% (Sahli). Dr. Maxwell said that ninety grammes of iron and ammonium citrate were administered each day to increase the formation of hæmoglobin.

At this stage, owing to some misunderstanding, the patient ceased to attend at the Royal Melbourne Hospital and had not reported again until April 9, 1937, when he complained of weakness and dyspnœa, and exhibited extreme pallor. A blood examination revealed a recurrence of gross anæmia, and "Campolon" injections had been commenced again on April 9, 1937, with a prompt response to treatment. Fifteen cubic centimetres of "Campolon" had been administered between April 9 and April 23, in three injections, and the red blood cells had risen from 1,730,000 to 3,240,000 cells per cubic millimetre, the hæmoglobin from 33% to 58% (Sahli), and the colour index had changed from 1·17 to 1·09. Dr. Maxwell said that intramuscular injections of five cubic centimetres of "Campolon" would be continued at weekly intervals until the blood picture was restored to normal, and then a maintenance dose (probably five cubic centimetres) would be administered once a month.

Dr. Maxwell considered that the lesson to be learned from this case was that injections of liver extract did not cure pernicious anæmia and that a relapse would

occur if the patient discontinued treatment; an adequate maintenance dose of liver extract was essential if a normal blood picture was to be maintained; desiccated stomach might be used if desired instead of liver therapy.

Gout.

Another patient shown by Dr. Maxwell was a male, fifty-one years of age, who twenty-three years previously had experienced sudden acute pain in the metatarsophalangeal joint of the right big toe. Since then he had had many attacks of pain in the elbow, wrists, hands, knees and ankles, with a gradual deposit of sodium urate in the form of very large tophi. In 1930 the patient came under observation at the hospital, complaining of pain in the epigastrium, which occurred some two hours after meals; there was also a history of melena. Radiographic examination did not reveal any organic lesion in the stomach or the duodenum, and his gout had been treated with *Vinum Colchici*, with relief of pain in the joints.

Dr. Maxwell said that between 1930 and 1937 the patient had had three attacks of hæmaturia, and in 1933 an ulcer of the lesser curvature of the stomach had been suggested by the radiographic appearances; this was not confirmed and some deformity of the duodenal cap indicated a lesion in this region, but the evidence was not conclusive. In April, 1937, the patient had had moderately severe hæmaturia and was given a transfusion of blood, and glucose with saline solution was injected intravenously. The estimations of the blood urea were: on April 24, 107 milligrammes; April 25, 131 milligrammes; April 30, 139 milligrammes; and May 16, 134 milligrammes per hundred cubic centimetres of blood. The uric acid ranged from 6·0 to 9·0 milligrammes per hundred cubic centimetres of blood. Following administration of "Atophan" the uric acid content of the blood fell from nine to three milligrammes per hundred cubic centimetres, but no effect on the blood urea was observed. A fractional test meal revealed a maximum free hydrochloric acid of 42 units and a total acidity of 54 units. The diastolic blood pressure was 110 and the systolic pressure 170 cubic millimetres of mercury. The patient was, at the time of the meeting, receiving an almost purin-free "ulcer type" of diet, with abundance of eggs and milk; one drachm of magnesium trisilicate was administered every four hours, and colchicum if the pain of the gout was severe. Dr. Maxwell remarked that, judging by the persistently raised blood urea, there was a considerable degree of renal insufficiency, but this was not caused by excessive administration of alkali powder, as the high blood urea still persisted on discontinuance of the alkali powder and substitution of magnesium trisilicate.

Melanoma of the Forehead.

DR. KAYE SCOTT showed a woman, seventy-nine years of age, who had reported three years before with an ulcerating melanoma situated over the glabella. This was treated with radon needles buried under, and wide of, the obviously involved area. The tumour mass slowly subsided over a period of three months, during which time the tumour gave place to granulation-like tissue of coal-black colour, over which the peripheral epithelium slowly grew. The final result showed a pigmented scar, the colour of a blue-black tattoo mark, which completely lacked any subjacent induration. No glands had become involved.

In discussion, Dr. Kaye Scott said that he feared surgical attack on obviously malignant melanomata, so much so that in this case not even a preliminary section had been taken, but stereoscopic photographs clearly showed the nature of the lesion at the commencement of treatment. These tumours should be well treated by well-screened interstitial radium, as large a dose being given as the tissues would tolerate. This form of treatment was much superior in its results to external radium applications, possibly because of the effect of the zone of intense radiation immediately around the needles. If the lesion failed to regress satisfactorily, or if healing processes failed, excision could then be considered, but was rarely found to be necessary. A feeling existed that surgical interference before radiation predisposed to glandular metastases. Dr. Kaye Scott remarked that during the years 1929 to 1934 eleven patients with melanomata, classed Stages I to III, had been treated at the Royal Melbourne Hospital with surgical excision in addition to radiotherapy; six patients only had survived. From 1930 to 1934 seventeen patients in similar stages had been treated with interstitial radiotherapy only; sixteen were alive and well.

Familial Epithelial Odontomes in Two Brothers.

Dr. Kaye Scott also showed two brothers, aged eight and eleven years, both of whom had developed bilateral epithelial odontomata. The older boy had received deep X ray therapy during the past five years, and recent skiagrams showed general filling of the absorbed areas. The lesion on one side had been curetted, but had progressed in spite of this treatment; therapy was given thereafter only to the surgically treated side. Only in the present year it was found that the younger boy was suffering from identical lesions, for which he was receiving treatment. A sister was unaffected, and no other relatives were known to be affected.

Neoplasms at the Corneo-Scleral Margin.

Dr. Kaye Scott also showed three patients with neoplasms at the corneo-scleral margin. One was a girl, eleven years of age, who at birth had had a pigmented mark at this site, which progressed till, when she was eighteen months old, it was excised. Six years later a recurrence appeared, the lesion being three to four millimetres in diameter and considerably raised. All the obvious tumour had disappeared at the time of the meeting following treatment three years earlier with an interstitial needle, and only a faint trace of pigment had remained.

An epithelioma at the same site in a man, sixty-two years of age, was also shown. This lesion had completely disappeared, leaving an apparently normal conjunctiva and unaffected sight. The patient was well three years afterwards.

Another patient shown by Dr. Kaye Scott was a boy, fourteen years of age, who had bilateral epitheliomata situated at the medial margins of the corneo-sclerotic junction. The macroscopic appearances had been confirmed by biopsy. Each lesion was four to five millimetres in diameter, and these lesions had disappeared under recent interstitial radon treatment.

Dr. Kaye Scott said that the patients had been shown to illustrate the technique used in these cases. Special radon needles of 0·5 millimetre of platinum equivalent screenage, made of gold to give minimum thinness, were threaded with a single thread at each end, which was let into the lumen of the needle to avoid having an eyed needle. The needles were bent to the curve of the outer curvature of the cornea; one thread was threaded to a cutting edge needle of similar curvature, which was introduced above the lesion, run round under the conjunctiva, over the lesion and out below the lesion; the needle was pulled into position by the thread and guided at the same time by forceps on the free end. The threads at each end were finally used to anchor the needles *in situ*. A series of five or six cases had been satisfactorily treated by this method without impairment of sight or permanent injury to eye structures.

Laryngeal Neoplasms.

Dr. Kaye Scott also showed two patients with laryngeal neoplasms.

An intrinsic carcinoma involving the greater part of the right true cord and the anterior commissure had been treated five years earlier after reflection of the infrahyoid muscles, removal of the whole of the right thyreoid cartilage and the anterior two-thirds of the left cartilage; the deep layer of perichondrium was left intact and over its surface radon needles were sutured. The cord showed irregularities corresponding to the previously ulcerated areas, but moved freely.

The other patient was a male, thirty-two years of age, who had three years earlier complained of laryngeal

obstructive symptoms, hoarseness and swelling over the larynx. A chondrosarcoma was found eroding the right thyreoid cartilage, which was removed with part of the growth in an attempt to "decompress" the larynx. Tracheotomy was performed, and in the following eighteen months five courses of intensive deep X ray therapy were administered. Dr. Kaye Scott remarked that, at the time of the meeting there was no mass to be felt and the larynx appeared relatively normal, without redness or swelling. The patient was well, having gained two stone in weight, and the tracheotomy had been allowed to close.

Atypical Polycythæmia.

DR. S. O. COWEN showed a patient, fifty-one years of age, who had had a toxic goitre fifteen years before, which had been treated medically, until she had had a subtotal thyreoidectomy done six months before the meeting. She had noticed a lump in her abdomen for two years, which used to slide up under the left costal margin and could not be felt. The lump was freely movable, and sometimes gave a sensation of stretching inside her abdomen; it had become larger and definitely firmer in the last six months. On examination of the blood the red blood cells numbered 8,450,000 and the leucocytes 14,650 per cubic millimetre, the colour index was 0·79, and the hæmoglobin value had been estimated at 134% (14 gramme standard). The red cells showed no abnormal morphology; the reticulocytes had been less than 1% and the white cells were of normal appearance. The systolic blood pressure was 184 and the diastolic pressure 106 millimetres of mercury.

Pernicious Anæmia.

Dr. Cowen also showed a male patient, fifty-eight years of age, who in February, 1937, had developed a pain across his chest on walking a hundred yards. The pain was not very severe; it was precordial and below the right nipple and in the region of both biceps. When the pain came on, the patient had to stop for three or four minutes and then it disappeared. There was no distressing degree of dyspnœa and no palpitation or sore tongue or abnormal sensation, but his complexion was noticed to be lemon yellow in colour. On April 5, 1937, when admitted to hospital, he could walk only fifty yards before the pain came on. The pulse was regular; the systolic blood pressure was 110, the diastolic pressure 66 millimetres of mercury. The apex beat of the heart was in the fifth intercostal space 10·0 centimetres (four inches) from the mid-line. The sounds were regular, but a systolic murmur was audible, maximal at the apex. The hæmoglobin value was estimated at 44% on the 14 gramme standard; the red cells numbered 2,050,000 per cubic millimetre; the colour index was 1·1. The patient's serum had failed to yield the Wassermann reaction. Dr. Cowen said that he had treated the patient by the administration of "Luminal" in doses of half a grain three times a day, and of iron and ammonium citrate in doses of thirty grains three times a day. Injections of "Campolon" were given on April 6, 17, 27, and on May 6, 1937, a start being made with a dose of 20 cubic centimetres, the dose being decreased to 15 cubic centimetres per day. The reticulocytes were less than 1% on April 7 and April 8, but increased on April 9 to 3·6%, and by April 10 they had reached 25%. A blood count, made on April 30, showed the hæmoglobin to have risen to 85% (14 gramme standard); the red cells numbered 4,730,000 per cubic millimetre, and the colour index was 0·9. On May 4, 1937, the patient walked approximately one and a quarter miles without pain or distress, and the pulse rate rose from 76 to 90 beats per minute.

Anaphylactoid Purpura.

Another patient shown by Dr. Cowen was a male, fifty-seven years of age, who complained that in July, 1936, the right knee had become swollen and stiff and he had had some abdominal pain. Four days later he had developed diarrhœa and passed some blood by the rectum. Three weeks later the swelling of the right knee had subsided and the left knee and hand had become swollen; there was some vomiting, and a few days later spots appeared on both legs and some swelling of the ankles and legs occurred. Since that time the patient had had continual crops of purpuric spots on both legs, with œdema of varying degrees. The patient said that he was a naval man and had been in the tropics with the Navy. Dr. Cowen pointed out that all investigations, including the culture of fæces, a Wassermann test and a test meal, had yielded negative results. The bleeding time, coagulation time and clot retraction had also been normal. Skiagrams of the chest and teeth revealed no abnormality; prostatic secretion on culture and microscopic examination had given negative results. The patient had been on a diet with high vitamin *C* content and he had had a course of intramuscular injections of sterile defatted milk, and on two occasions he had had transfusions of ten ounces of blood.

Macrocytic Anæmia.

Dr. Cowen also showed a patient who was suffering from macrocytic anæmia following partial gastrectomy for pyloric carcinoma. The patient was a man, seventy-five years of age, who had been admitted to hospital on February 26, 1937. Pyloric carcinoma had been proved by section and removed by partial gastrectomy in April, 1934. In January, 1936, the macrocytic anæmia had been much improved by the use of "Campolon", but the patient had lost condition and had no appetite, the tongue was sore, and he had been sleeping badly since he was last in hospital. He was much paler; he became dyspnœic on walking about fifty yards; and he had no palpitation or cough; the œdema of the ankles had subsided. There were no changes in the finger nails, and no dysphagia, though the patient had lost two stone two pounds in weight in approximately two months. Blood examination on February 26, 1937, showed the hæmoglobin value to be 31% (14 gramme standard); the red cells numbered 1,410,000 and the leucocytes 1,450 per cubic millimetre; the colour index was 1·1. Gross anisocytosis and poikilocytosis were present. No nucleated red cells were seen and the reticulocytes were less than 1%. The patient was given "Campolon", a start being made with an initial dose of five cubic centimetres on February 26, 1937; this was increased to ten cubic centimetres on February 27 and 28; on March 13 he received an injection of 20 cubic centimetres and another on March 31, and he had a final dose of ten cubic centimetres on April 7, 1937. The reticulocytes rose from 3·8% on March 1 to 28% on March 3, 1937. Blood examination on April 6, 1937, showed the hæmoglobin value to be 62%; the red cells numbered 3,305,000 per cubic millimetre; and the colour index had dropped to 0·94.

Thrombo-Angiitis Obliterans Treated by Sympathectomy.

DR. A. E. COATES showed a male patient, thirty-five years of age, who complained that for two years he had suffered with very cold feet during the winter; in the summer they became hot and swollen and sweated freely; they were constantly painful while he was resting. For twelve months prior to his admission to hospital he had noticed a red blotch about half way down on the anterior surface of the right leg; this spot was very painful, the pain spreading all over the leg; six months later a similar blotch appeared on the left leg. The patient had said that two months before his admission, when he was working on concrete, the toes of the right foot became numb and the sole of the foot was tender; the sweating of both feet became very pronounced and the skin was macerated. On examination of the right leg no pulse was palpable in the anterior tibial, posterior tibial or popliteal arteries; the femoral pulse was palpable. The leg became cold and pale on elevation, and red on depression—the typical rubor of vascular occlusion. In the left leg all pulses were palpable except that of the posterior tibial artery. The left was warmer than the right leg, and had slight colour changes on being postured. There was no ulceration or gangrene. Prior to the patient's admission to hospital thermal tests had been carried out under

spinal anæsthesia, two cubic centimetres of "Ethocaine" being used. Dr. Coates demonstrated the results of the thermopile tests diagrammatically; the charts for the first toe, the fifth toe and the sole of the foot showed a constant rise for the left leg as compared with the right.

Bilateral lumbar sympathectomy and appendicectomy were performed on July 10, 1937, by the transperitoneal route, and on July 30 the patient was discharged from hospital with both feet warm. He had survived the previous winter and had been very comfortable during the cold months of this year up to the date of the meeting. His feet remained warm and he was able to work. Dr. Coates said that the condition was an early stage of Buerger's disease in which the vasospastic element was pronounced.

Spinal Tumour Meningioma.

Another patient shown by Dr. Coates was a married woman, thirty years of age, who had been admitted to hospital on May 13, 1936. Six months earlier the patient had noticed that the bed-clothes tickled her legs; two months afterwards she had for a period of three weeks had difficulty in starting micturition. There was a numbness and tinkling in the legs which gradually spread to the lower part of the abdomen; her feet became clumsy and felt stiff. Two months before her admission to hospital she had developed a staggering gait and would fall over suddenly without warning; two weeks later she complained of a sharp pain in the small of her back, at the level of about the seventh or eighth thoracic vertebra, and a few weeks later she collapsed and was able to walk only with difficulty. Examination of the central nervous system had shown that the power in the lower limbs was diminished; the deep reflexes were hyperactive, more on the left than on the right side; a bilateral flexor plantar reflex was present; the superficial abdominal reflexes were absent. There was loss of pin-prick sensation up to the tenth thoracic level; loss of vibration sensation was noted up to the third lumbar level and sensation to cotton wool was diminished at the eighth thoracic level anteriorly and posteriorly. The cranial nerves and the pupils were normal. The cerebro-spinal fluid was normal both chemically and microscopically; the pressure was 180 millimetres; a normal reaction was obtained to pressure on the jugular vein. The Wassermann and colloidal gold tests gave normal results; the fundi were normal. The blood film and skiagram of the spine were also normal. A lipiodol examination revealed a partial block at the fifth thoracic vertebra. On June 3, 1936, operation was performed under ether anæsthesia, the intratracheal method being used, and a small pinkish-white, rounded tumour, half an inch in diameter, was found at the level of the fifth thoracic vertebra, which on microsection was found to be a psammoma. The patient suffered from incontinence of urine and fæces after the operation. On June 30 the tone in the right leg was normal; the reflexes were equal; the plantar reflexes were flexor and the sensation to pin-prick and cotton wool was normal. At the time of the meeting the patient was quite well and had resumed the teaching of dancing.

Trigeminal Neuralgia and Carcinoma of the Stomach.

Dr. Coates showed a male patient, seventy-five years of age, who had been admitted to hospital on March 9, 1937. For three years he had suffered from attacks of severe stabbing pain in the right cheek, always beginning under the right malar prominence; the attacks were worse at night and seemed to be precipitated by cold weather and draughts. The attacks had become worse until he was afraid to eat. He lost weight and had had all his teeth taken out. Salivation and lachrymation had accompanied the attacks of pain.

On examination the patient was found to have tenderness under the right malar prominence, where slight reddening of the skin appeared; there was no tenderness over the supraorbital, infraorbital or mental foramina; the "trigger area" was in the naso-labial fold. On March 10 the right Gasserian ganglion was injected with absolute alcohol under local anæsthesia, and the next day complete anæsthesia to light touch was present from the lower margin of the orbit to below the angle of the mouth on the right side; the right eye was reddened and herpes had appeared on the upper lip. On March 13 the upper lip became swollen and there were vesicles inside the mouth, but there was no pain and the patient was discharged from hospital. The lower hemisphere of the eye remained red, the upper half of the globe being normal. Four weeks later the patient had anorexia and a feeling of obstruction to the passage of food in the xiphisternal region. A skiagram was taken and the presence of a large carcinoma of the stomach was reported.

Dr. Coates said that up to the time of the meeting there had been no recurrence of the facial pain, and he thought that the case illustrated well the condition of paroxysmal facial tic and its relief by ganglion injection with absolute alcohol. The Härtel method had been employed, the ophthalmic fibres being spared, and an eye-shield was still worn to protect the lower half of the cornea, which was insensitive. Dr. Coates remarked that the carcinoma in this case was probably inoperable, but exploration was to be undertaken in the week following the meeting.

Carcinoma of the Stomach in a Diabetic Patient.

Dr. Coates also showed a woman, sixty-two years of age, who had been admitted to the hospital on April 23, 1937. She had been treated for *diabetes mellitus* for four years with a diet of 125 grammes of carbohydrate, 70 grammes of protein, and 75 grammes of fat; she had not had any insulin. Six weeks before her admission to hospital she had had anorexia and a sense of heaviness in the stomach, and two weeks later had vomited copiously after a heavy meat meal; the vomiting, of whitish brown material, containing no blood, had been repeated twice a week; the patient had taken very little solid food and she had lost half a stone in weight in a month. There was no pain and she had no previous history of dyspepsia. On examination there was no mass, no visible peristalsis was present, and the liver was not palpable. The presence of a neoplasm was suspected as a result of giving a barium meal; there was great distension of the stomach and gross pyloric obstruction. On April 24 the diet was changed to semi-solids taken six times a day. The patient had vomited the next day and the stomach was emptied by a Rehfuss tube and washed out; the continuous rectal administration of a 5% solution of glucose in saline solution was commenced. Two days later the blood sugar was estimated at 0·1%, and after preparation of the stomach partial gastrectomy was performed by the anterior Pólya method. A carcinoma obstructing the pylorus was removed; no liver secondaries were detected. On May 1, owing to the presence of a moderate amount of sugar and of diacetic acid in the urine, fifty units of insulin were given with fifty grammes of glucose; for eleven days ten units of insulin were given daily, and the diet was steadily increased to 150 grammes of carbohydrate, 67 grammes of fat and 70 grammes of protein.

Dr. Coates said that this was a fortunate example of a small local growth causing early obstruction of the pylorus and lending itself to easy removal; being a diabetic, the patient was operated on under local anæsthesia, with a light administration of nitrous oxide gas during the intra-abdominal manipulations.

Gastric Carcinoma.

Dr. Coates also showed a male patient, sixty-two years of age, upon whom he had operated for a gastric carcinoma. In 1919 the patient had had an operation for perforated duodenal ulcer with general peritonitis, and in 1927 an incisional hernia was repaired. In 1929 a profuse hæmatemesis and melena brought about his admission to hospital. Investigation had established the presence of a duodenal ulcer, for which he was treated medically. He was in hospital again in 1932 with acute colitis. For the present illness he had been admitted on November 5, 1936, with a history of burning upper abdominal pain, which had been present for two weeks and which was different from the ulcer pain; it had not been relieved by food or

alkalis. Though his appetite was good, he was nauseated and constipated, and had lost a little weight. A large tender resonant mass, moving with respiration, was felt in the upper part of the abdomen, and the liver dulness was increased to two fingers' breadth below the costal margin. A report on the barium meal was that a filling defect was present affecting the middle third of the body of the stomach; it was very suggestive of a neoplasm and the duodenal cap was deformed. At a test meal on November 26 achlorhydria was found with the presence of blood and mucus and lactic acid. A week later Dr. Coates had performed partial gastrectomy by the anterior Pólya method. A section of the growth proved it to be a small-celled carcinoma. The patient had vomited dark fluid for four days and had a transfusion of blood, subpectoral administration of saline solution and continuous aspiration of stomach contents. At operation the stomach was adherent to the left upper abdominal wall and diaphragm. At the time of the meeting the patient was perfectly well and was gaining weight. Dr. Coates considered that this case was of interest in that a condition of hyperchlorhydria with ulcer was later replaced by a condition of achlorhydria with carcinoma.

Fracture of the Radius.

Dr. Coates also showed a female patient, sixteen years of age, who had been admitted to hospital on May 6. Five days earlier she had fallen off a bicycle on to her outstretched left hand. On her admission to hospital the left wrist was swollen, bruised and tender over the volar aspect and the dorsum, and all movements at the wrist were limited; there was an abnormal bony prominence on the volar aspect of the radio-carpal joint. The skiagram indicated that one small volar fragment was rotated through 180° and the outer fragment was displaced posteriorly, taking the carpus with it. The arm was plastered in full volar flexion, but in a subsequent film it could be seen that the position was not improved. On May 14 Dr. Coates had operated and removed through a longitudinal volar incision the small malplaced fragment and had applied anterior and posterior plaster slabs to the elbow with the wrist in full volar flexion. Two days later the plaster was extended to include the elbow.

Dermatological Cases.

DR. ROLAND R. WETTENHALL showed a number of patients with dermatological conditions, including the following.

Multiple Keratoses.—A woman, eighty years of age, with multiple keratoses of many years' duration, showed the development of rodent ulcers in several areas. The general atrophic condition of the skin had improved after three months' treatment with a mixture containing iron and ammonium citrate (ten grains) and *Liquor Arsenicalis* (two minims) given three times a day, and many keratoses had been considerably reduced by quinine cream (A.P.F.), applied at night time, and a lotion composed of three drachms of *Oleum Ricini* made up to two ounces with rectified spirit, which was dabbed on in the day time. A superficial radium plaque was being applied to the more prominent lesions.

Basal-Celled Carcinoma.—A man, sixty-nine years of age, showed basal-celled carcinoma of the nodular variety, of two years' duration, on the side of the neck. This was a firm, raised, vascular nodule, not deeply infiltrating, of a type which cleared readily with superficial radium therapy, in which approximately 5% of the β rays were used.

Tertiary Syphilide.—A man, fifty-five years of age, had been seen at the hospital five months previously with a large ulcerating gumma of the upper lip and other signs of tertiary syphilis. At that time a section of the lesion was reported as an early epithelioma, but after twelve injections of "Bismol", at an interval of one week, in doses of two cubic centimetres, it had healed completely. The Wassermann test gave a strongly positive reaction, twenty-four minimal hæmolytic doses of complement being fixed, and anti-syphilitic treatment was being continued.

Infective Granulomata.—A man, seventy-five years of age, suffered from painless, elongated, pustular, granulomatous lesions on both forearms of twelve months' duration following a papular eruption. There was also a follicular oozing and crusted eruption on the dorsum of both feet and ankles. There was no gross enlargement of the axillary or inguinal lymph glands. The Wassermann test yielded no reaction. Dr. Wettenhall had considered either sporotrichosis or blastomycosis as ætiological factors, but culture of pus from the lesions had yielded only *Staphylococcus aureus.*

Lichen Planus.—A woman, forty-eight years of age, was shown with acute *lichen planus* of three weeks' duration. A scattered, violaceous, waxy, papular eruption was present chiefly on the extremities, and thick white plaques were present on the mucous membranes of the cheeks. There had been no apparent external cause and the patient's general health was good. Treatment consisted in the administration of *Mistura Liquor Hydrargyri Perchloridi*, one drachm being given three times a day. Local applications of *Lotio Calamini* with *Liquor Carbonis Detergens* were also used.

Herpetiform Dermatitis.—A man, twenty-nine years of age, had suffered from recurring attacks of herpetiform dermatitis for fifteen months. Each attack had responded readily to *Liquor Arsenicalis* (five minims three times a day), but had recurred within a fortnight each time the mixture was discontinued. The condition was also much aggravated while the patient was working at a silk stocking factory in a hot, moist atmosphere, but the ordinary cutaneous tests for sensitivity to the materials used had yielded no reactions.

Routine Methods in the Investigation of Blood Diseases.

DR. HILDA GARDNER and DR. IAN WOOD gave a demonstration of routine methods used in the diagnosis and treatment of the diseases of the blood. It was shown that the Tallquist hæmoglobin scale was inaccurate, especially when a specimen of blood with very low hæmoglobin content was being tested. It was suggested that an instrument such as the Sahli model should be employed and that it would yield a greater degree of accuracy.

In the study of iron deficiency anæmia it was important to consider the hæmoglobin percentage rather than the red cell count, as the red cells were often at quite a high level, counts of four million or more being quite common. Large doses of iron were necessary to effect a cure in these cases, and liver extracts and stomach preparations were valueless. In severe cases the normal hæmoglobin content might not be restored until the treatment had been maintained for two or three months.

In the treatment of pernicious anæmia it was best to give a mixed form of therapy, consisting of whole liver on one or two days a week, and a potent dry stomach extract, such as "Ventriculin" on two days a week, and to maintain a hæmoglobin percentage of over ninety by means of intramuscular injections. Highly concentrated preparations of liver for intramuscular injections, such as "Anahæmin" (British Drug Houses), were now available, and with a mixed form of treatment injections needed to be given only every one or two months, according to the individual reaction of the patient.

The typical cells seen in glandular fever (infectious mononucleosis) were demonstrated, and it was pointed out that there was some doubt as to whether these cells belonged to the monocytic or lymphocytic series. Paul and Brunnell's test for this disease was mentioned. The profuse reticulocytosis of acholuric jaundice was demonstrated and the significance of the corpuscular fragility test in the diagnosis of this disease was discussed. The importance of employing a suitable brand of cresol blue stain for reticulocytes, especially in the dry method, was shown; very satisfactory results had been obtained with the British Drug Houses preparation. Films showing the disappearance of the polymorphonuclear cells of the body in agranulocytosis were also demonstrated, and special reference was made to the hypersensitivity of some persons to drugs of the amidopyrine group. Dr. Wood said that X ray therapy played an important part in the control of

cases of chronic leuchæmia and had often prolonged life for eight years or more. Moreover, the clinical condition was greatly improved by intensive iron therapy when the hæmoglobin was found to be at a low level.

(To be continued.)

VICTORIAN BRANCH NEWS.

Fees for Medical Services Rendered at the Request of Government Departments.

THE following three cases have been referred to the Branch Council by members in recent months.

At the request of the Secretary of the Transport Board, a consulting surgeon visited a town 180 miles from Melbourne to advise and consult with a doctor attending an officer of the Board seriously injured in an accident. Prior to his departure the surgeon was advised that "the Government would bear all expenses in connexion with the visit". On his return he reported to the Secretary and the Chairman of the Board, and later rendered his account. Payment was refused by the Government on the ground that: "The expense was apparently incurred at the instance of an official not authorized to bind the Crown in contract."

In the second case a doctor was called by the Police Wireless Patrol to inspect the body of a man found shot. The Police Department disclaimed responsibility for his fee and advised him to collect it from the relatives.

In the third case a doctor, at police request, travelled at night time a distance of thirteen miles to a neighbouring town to certify an insane person. In return for his services the Mental Hygiene Department paid him one guinea for certification and sixpence a mile travelling expenses.

What, then, is the real position? The surgeon in the first case quoted has no redress except against the patient or the Secretary of the Transport Board. The Chief Commissioner of Police has been interviewed regarding the second and third cases, and he states that: (i) When a doctor is called at the request of the police for police purposes the department will accept responsibility and the doctor in case two will almost certainly be paid. (ii) In case three the police acted as agents for the Mental Hygiene Department and that department is responsible for fees under its own scale. (iii) In all cases members called by the police should, before answering such calls, ascertain whether they are being called at "police request" or whether the police are merely acting as intermediaries for some third party.

Police regulations lay down the following fees in cases where the department accepts responsibility: for cases attended between 7 a.m. and 9 p.m., 15s.; for cases attended between 9 p.m. and 7 a.m., £1 11s. 6d. Mileage by motor car is 3s. 6d. per mile, one way, after the first mile.

Despite prolonged efforts, it has not been possible to ascertain what Government departments or statutory bodies are so constituted as to be responsible for expenses incurred by the act of their servants in engaging the services of a doctor, and members are advised to ascertain their position definitely before attending cases in such circumstances.

"Progressive" Lodge Certificates.

Following representations from the Friendly Societies' Association, the Branch Council has consented to request members to supply, when required, the first progressive certificate at the end of one week, instead of two weeks. The friendly societies seek this concession to safeguard their sick funds.

Following inquiry, a member was informed that when a lodge member was an inmate of a public hospital and not under his care he could not fill in the standard "progressive certificate", but that his certificate should take the following form: "I last saw Mr. ..... on ..... and am informed that he is still in hospital."

X Ray Equipment.

Information has been received from the Honorary Secretary of the Victorian Branch of the Australian and New Zealand Association of Radiology that an advisory committee has been formed to advise on the purchase, installation *et cetera* of new radiological equipment. Any member or hospital committee seeking the help of this committee should communicate with the convener, Dr. Alan Mackay, Parliament Place, C.2.

International Conference on Education.

The International Conference on Education, under the auspices of the New Education Fellowship, England, will meet at Melbourne from August 24 to August 31, 1937. The membership fee is one guinea. The programme contains much of interest to medical men and may be inspected at the Branch office. Will those wishing to attend please notify the Medical Secretary and remit the fee.

Mallee Doctors.

The following letter has been received from the Secretary of the Closer Settlement Commission:

3rd July, 1937.

With reference to the arrangements set out in letter to you dated 16/10/35, respecting payments to Mallee doctors in the cases of settlers under lien to the Commission, I have to inform you that, as from the 1st July, 1937, the Commission will cease to make advances. I have to add, therefore, that as the Commission will not control harvest returns of next season's crop, no responsibility can be taken by it respecting the indebtedness of its settlers to doctors in the future.

Warning Notice.

Members of the Victorian Branch are warned against a woman apparently suffering from asthma, who endeavours to obtain injections of morphine by falsely representing that she is a patient of a consulting physician who has been giving her the drug.

Congratulations.

The Branch Council has extended its congratulations to Dr. J. P. Major, on whom His Majesty the King has conferred the honour of Commander of the Most Excellent Order of the British Empire.

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the Western Australian Branch of the British Medical Association:

Gray, William Blair Christie, M.B., B.S., 1937 (Univ. Melbourne), Perth Hospital, Perth.

The undermentioned have been elected members of the Victorian Branch of the British Medical Association:

Hayes, Horace Townsend, M.B., B.S., 1936 (Univ. Adelaide), 60, Howitt Street, Caulfield.

Hogg, James Edgar Phipps, M.B., B.S., 1930 (Univ. Sydney), A.M.P. Buildings, Bendigo.

Stephens, Frank Douglas, M.B., B.S., 1936 (Univ. Melbourne), Royal Melbourne Hospital, Lonsdale Street, Melbourne.

Post-Graduate Work.

MELBOURNE PERMANENT POST-GRADUATE COMMITTEE.

Primary Fellowship Examination of the Royal College of Surgeons of England.

THE Melbourne Permanent Post-Graduate Committee announces that a course of instruction for the Primary Fellowship Examination of the Royal College of Surgeons of England will commence in Melbourne on Monday, July 26, 1937, and will continue until shortly before the examination on November 29, 1937.

The lectures in anatomy will be given at the Anatomy School, University of Melbourne, on Monday, Wednesday and Friday, at 5 p.m., by Professor F. Wood Jones, assisted by Dr. S. Sunderland.

The lectures in physiology will be given on Tuesday and Thursday at 4.30 p.m., and on some Saturdays at 10 a.m., at the Walter and Eliza Hall Institute, Royal Melbourne Hospital, by Dr. C. H. Kellaway, assisted by Dr. J. I. Hayward.

The fee for the complete course will be twenty guineas, and for students ten guineas. Prospective candidates must notify the Honorary Secretary of the Melbourne Permanent Post-Graduate Committee, 33, Collins Street, Melbourne, as soon as possible. A cheque for the fee must be enclosed with this notification.

Candidates are reminded that it is necessary to enter for the examination on or before July 31, 1937. Entrance forms are obtainable from the Secretary of the Melbourne Permanent Post-Graduate Committee, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

Obituary.

DONALD ALLAN CAMERON.

WE are indebted to Dr. W. N. Robertson for the following account of the career of the late Dr. Donald Allan Cameron.

Donald Allan Cameron was born at Ipswich, Queensland, in 1878. He was the son of Donald Cameron, the much loved headmaster of the Grammar School. Donald was a good scholar and played good cricket and tennis. He was always of the plodding, steady, working type, and continued to be so during his 'varsity life, and indeed all his life. He graduated as Bachelor of Medicine in 1900 and Master of Surgery in 1901, and for two years was resident medical officer at the Sydney Hospital. He first joined his brother, J. A. Cameron, in Ipswich in general practice, eventually coming to Brisbane, where he gradually built up a wide and high class practice. He was appointed to the staff of the Hospital for Sick Children and Lady Lamington Hospital for Women, where his meticulously careful and devoted work secured the admiration and confidence of his fellows. He developed a flair for surgery, and it was his extreme care in preparation, operative skill and after-care that made his surgery so valuable and successful. Furthermore, he was absolutely honest in his relations with his patients and his colleagues.

When war came, he went overseas as a major and he became the head of the surgical unit at Harefield. Later he went to France and Belgium as head of a surgical unit at Remy Siding. There his association with the mixed units of Canadians, Americans and British surgeons seemed to be the happiest and most stimulating experience in his life. He was recalled to take charge of the orthopædic work at the Rosemount Hospital, after acquiring orthopædic experience in Paris and Liverpool. He resigned from that position, as he found the lack of equipment and support rendered the work distasteful.

He continued his work at the Lamington Hospital and the Children's Hospital until his appointment expired.

He was Secretary of the Queensland Branch of the British Medical Association for a time, and also President. He performed the duties of these offices with the same methodical care that characterized all his work. He was rightly made a foundation Fellow of the Royal Australasian College of Surgeons, as his life and work were typical of the ideals of the college. It is consequently easy to give an estimate of his work and character. He lived up to all that is highest in his profession, and was the staunchest of friends.

For the last two years he steadily failed in health, but bore his affliction with the greatest fortitude. His only complaint was that he was "so much trouble" to his wife and his friends. His wife nursed him devotedly night and day, and he was surrounded by such loving care that he was prevented from feeling his steady decline. To those of us who were bound to him by deep affection and regard, his passing has left a very painful blank. He lived nobly and was a very real example of the nobility of our profession.

Dr. G. P. Dixon writes:

The untimely death of Donald A. Cameron is a tragedy. His unfailing courtesy and kindliness had endeared him to a wide circle of friends, while his skill and care will cause him to be remembered and mourned sincerely and regretfully by his numerous patients.

His end was tragic in that he was forced to cease work while a comparatively young man and to face a lingering, painful illness without the solace of his one hobby—work. Not even the loving kindness and unceasing devoted care of his wife and relatives could quite make him forget the loss of this work. A more single-minded man never lived; his patients and his profession were always his chief and, if necessary, his only consideration.

Educated at the Ipswich Grammar School and Sydney University, his scholastic career was characterized by industry, thoroughness and pertinacity in getting things "just right". These qualities he retained all through his professional life. After service in residence at the Sydney Hospital he entered private practice at Ipswich in conjunction with his brother, Dr. J. A. Cameron. There he worked hard and learned a lot about general practice from his brother, of whom he always spoke in terms of sincere affection and admiration. Thence he went to Brisbane, still in general practice, building up the edifice of his surgical specialty on the foundation so well and truly laid in Ipswich.

His appointment to the honorary staffs of the Hospital for Sick Children and Lady Lamington Hospital for Women gave him scope for his undoubted surgical ability and coloured the whole of his future professional activities. Although he was a general surgeon with a very broad outlook, the surgery of childhood and gynæcology showed him at his best.

Donald Cameron volunteered for active service in spite of ill-health, and his surgery at Harefield Hospital and later on the French front with a Canadian casualty clearing station has been described as excellent by those who came in contact with him there. Many grateful soldiers still sing his praises.

Later he was recalled to Australia, much against his will, and placed in charge of the orthopædic military work in Brisbane. The disorder and "red-tape" regulations existing at that time did not conform to his ideas of what should be, so he decided to resume civilian practice as a surgeon. This period probably represented the zenith of his career. He soon established a sound, extensive practice as a consultant surgeon. The fine quality of his judgement and craftsmanship can be gauged by the confidence he inspired in his colleagues, numbers of whom, with their families, became his patients and found their confidence justified.

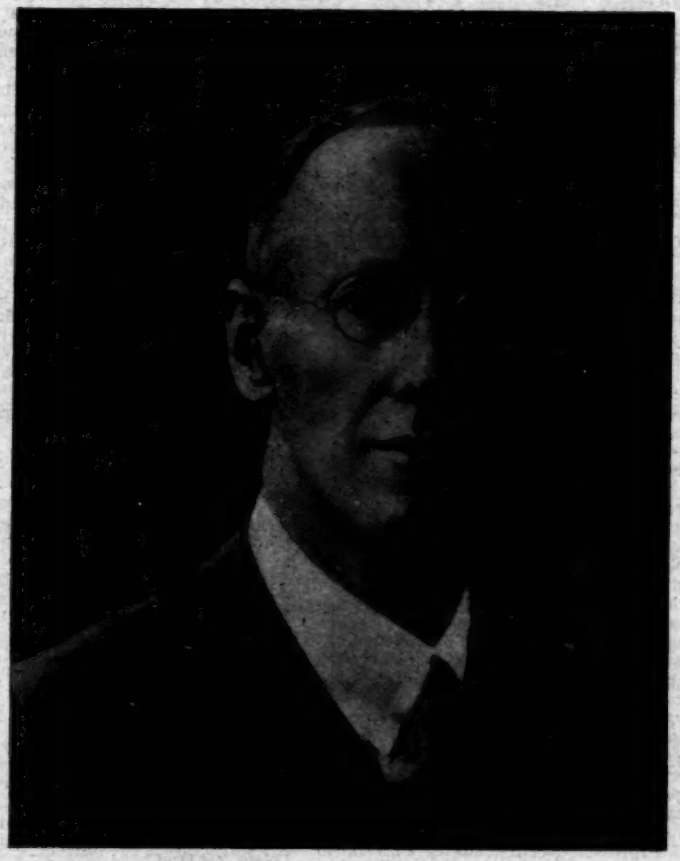
The younger men and women who had served as residents under him at the public hospitals were particularly grateful to him for help and guidance in their troubles and difficulties. They called him in consultation and sent

him many surgical cases, knowing that he would play the game on all occasions and give his best both to patient and medical *confrère*. One could not wish to work with a more helpful and considerate colleague.

Dr. Cameron did not write much; the few papers he read at meetings were of a very high order and showed originality of outlook combined with sound practical knowledge of his subject, and therefore were always very welcome to his listeners.

He was always ready and willing to help the nurses in training and devoted a lot of his time to giving unofficial demonstrations and lecturettes, which they found extremely useful.

His meticulous care in diagnosis and treatment, combined with gentleness and thoroughly sound judgement, ensured the excellent results which he certainly achieved. He never attempted "fireworks", saying that, in the interests of his patients, he found deliberate thoroughness and gentleness to be the safest and best procedure.



His ethical standards were always of the highest order—absolutely straight and unswerving. His patient came first, other considerations nowhere.

It is to be hoped that his splendid work for the medical profession done while he was Honorary Secretary of the Queensland Branch and later as President of the Branch and member of committees, will be commemorated by men who were associated with him at that time.

A fitting recognition of his high ideals and professional standing was his invitation to become one of the founders of the Royal Australasian College of Surgeons. He was keenly interested in the formation and growth of this college until his failing health forced him to retire.

We who enjoyed the privilege of his friendship will miss him sorely—such staunch friends are very rare—but we shall always be proud to recall his splendid achievements in his chosen field of labour—in many cases a labour of love, without hope or wish for reward, and done with the handicap of a chronic, painful illness which would have beaten most men. Also shall we gratefully remember his intolerance of any form of chicanery, his dogged perseverance and patience in seeking the truth, his absolute integrity, his unfailing courtesy and kindliness, and his wonderful gentleness and generosity. Some such words could fittingly form part of the epitaph of Donald Cameron; and who could wish for better?

Dr. J. Lockhart Gibson writes:

Dr. Donald Allan Cameron died recently in Brisbane after a long and painful illness, which had laid him aside from work for the past two years. He combined many valuable and lovable qualities, qualities that enriched his profession and were an outstanding example of rectitude, gentleness and devotion to his work. He "worked for the love of the working". He lived and worked quietly, and was fortunate in an ideal home life. The public and his medical brethren valued and profited by his outstanding surgical acumen and skill. His work was characterized by extreme thoroughness. He never was satisfied with "that should be enough". A complete job had, if possible, to be made. He was ever anxious to keep his surgery up to date, and when on holiday in larger cities spent much of his time watching the work of others and in picking their brains.

Sir Alexander MacCormick was his ideal surgeon, and he enjoyed nothing better than watching him and profiting by his friendship. He was "Donald" to his friends, and there was an affectionate ring in the sounding of the word. We shall continue to miss him as a friend and as a colleague more than I can say.

Incidentally, Donald Cameron's father, the late Donald Cameron, M.A., said to me regarding his sons as they were starting their medical career: "I want them to be of some use in the world." They fulfilled his wish.

Dr. D. Gifford Croll writes:

I have known and worked with Donald Cameron for more than twenty-five years, but nothing that I can say will illustrate the quality of his surgery so well as the following incident.

I was called in the night to see a woman who had an attack of abdominal pain and had vomited once. She appeared to be a little shocked; her pulse rate was 120, her temperature was normal; she had slight tenderness and a little rigidity in the right lower region of the abdomen. Nothing abnormal was found on vaginal examination. I took her into a hospital and sent for Cameron. When he arrived about an hour later all of her symptoms had disappeared. She had no pain, no tenderness or rigidity, her pulse and temperature were normal, and she looked normal. She had been given an enema, which she retained. Cameron said he would operate, although he agreed with my suggestion that he might find nothing abnormal. Upon opening the abdomen the peritoneal cavity was found to be full of blood. He went straight for the right ovary and tube and brought out a tubal abortion, bleeding profusely. This he tied off and I then expected him to close up the abdomen. Instead of that he started upon a systematic search of the abdominal cavity, and from the left upper region he brought down a large volvulus involving about fourteen inches of bowel. Circulation appeared to have stopped, and he remarked: "I'm afraid she's done for; she'll never stand resection of this. How-

ever, there's a sporting chance that it may recover." He untwisted the volvulus, returned the bowel to the abdomen and closed it. The woman made an uninterrupted recovery and is well today.

When the operation was over I asked him two questions: (1) What is the relation between those two conditions, because I cannot believe that it was a coincidence? He replied: "No, it cannot be a coincidence. I think she must have had a tubal pregnancy and the volvulus then occurred and caused her to abort." (2) What made you search for the volvulus? Wasn't the tubal abortion enough? To which he replied: "A patient with a tubal abortion should not retain an enema."

SIMSON STUART.

WE regret to announce the death of Dr. Simson Stuart, which occurred on February 5, 1937, at Rockhampton, Queensland.

Correspondence.

MALARIA AND ITS TREATMENT BY THE GENERAL PRACTITIONER.

SIR: I am inclined to think that Dr. Calov overlooked what, unfortunately, appeared in my paper only as a footnote, namely, that my statements were extracted from Professor Nocht's "Malaria".

When an authority like Professor Nocht, after thirty years of intensive practice in the treatment of malaria with quinine; and being the author of the so-called "Nocht Quinine Cure", which, since 1904, has stood the test of time; it being the first-introduced systematic method of prescribing quinine, based on the exactness of a daily, restricted, dose, and the division of that dose; when such an authority is prepared, within a few years of the appearance (in 1930) of "Atebrin", to hand the palm to the new drug, it requires more evidence than an "I think" to challenge Professor Nocht's statements.

For instance, as stated in my paper, Professor Nocht in his publication (1936) begins the chapter, which details his method of prescribing quinine, with: "We recommend—if quinine is still to be used at all—the method introduced in the Hamburg Institute in 1904"; by which is clearly meant that, if the other drug is available, it should be given preference.

Dr. Calov, on the other hand, writes: "I think that people should know that quinine is still an important, if not the most important, drug in the treatment of malaria."

In reference to "Atebrin" and "Plasmoquin", Dr. Calov writes: "I think that people should know that "Plasmoquin" is not without its dangers, and that a cure with "Atebrin" is not so simple as it sounds."

In my paper I quoted Professor Nocht's advice that it is inadvisable to administer "Plasmoquin" by itself. I also quoted his statement that "Atebrin" and "Plasmoquin" should only be given when the administration of the latter follows immediately that of the former. To quote Professor Nocht: "This method cannot be too strongly recommended; it has been employed by us for years without any drawbacks to our patients."

In this combination of the drugs we have the nearest possible approach to a *therapia magna sterilisans*.

In the treatment of malaria, perhaps more than with any other disease, it is disastrous to work on the lines of "I think this", or "my opinion is that". What is essential is a devoted following in the footsteps of those great men who have blazed the trail.

Yours, etc.,

ALFRED E. FINCKH.

Sydney,
July 9, 1937.

THE SURGICAL TREATMENT OF SPASTIC PARALYSIS.

SIR: In reply to Dr. Kelly's criticism of my paper, "The Surgical Treatment of Spastic Paralysis", I have never seen any indication that the blood flows from the limb with the higher diastolic pressure to the limb with the lower diastolic pressure. The reason, that Dr. Kelly has forgotten, is that the pressure in the capillary bed and veins is less than the diastolic pressure in either the spastic or normal limb and may be as low as six millimetres of mercury when the capillaries are fully dilated. The flow is into the capillaries and not out of the arteries of the spastic to the other limbs.

The analogy that Dr. Kelly draws to the blacksmith's bellows suits my purpose admirably and I thank him for the illustration. The movements of the bellows represent the movements of the heart producing systolic pressure. The nozzle of the bellows represents the diastolic pressure and the air stream, gradually decreasing in velocity, represents the capillary bed. The oxygen is not carried past the glowing coke more rapidly, but the stream is retarded in its progress by this very obstruction. This represents the effect of arteriolar constriction and capillary dilatation.

I did not say that the arterioles and capillaries may be dilated at the same time, for I do not believe this is so, excepting in acute inflammation. When the arterioles are dilated the capillaries are contracted and *vice versa*.

There is no need to call in speculations regarding alterations in electrical conditions as an explanation of the effects of superior thoracic ganglionectomy when we have the simple explanation of the alteration of the blood supply. It is impossible to ascribe the contralateral loss of tone in the lower limbs to any factor other than acceleration of capillary flow, which has been proved by my experiments.

Yours, etc.,

185, Macquarie Street, Sydney,
July 12, 1937.

N. D. ROYLE.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION): FIFTH SESSION.

Office-Bearers of Congress.

THE following is a list of the principal office-bearers of congress:

Patrons:

His Excellency Brigadier-General the Right Honourable Alexander Hore Arkwright Baron Gowrie, V.C., K.C.M.G., C.B., D.S.O., Governor-General of the Commonwealth of Australia.

His Excellency the Right Honourable Viscount Galway, D.S.O., O.B.E., Governor-General of the Dominion of New Zealand.

His Excellency Colonel the Right Honourable Sir Leslie Wilson, G.C.S.I., G.C.I.E., C.M.G., D.S.O., Governor of Queensland.

His Excellency Captain the Right Honourable William Charles Baron Huntingfield, K.C.M.G., Governor of Victoria.

His Excellency the Honourable Sir James Mitchell, K.C.M.G., Lieutenant-Governor of Western Australia.

His Excellency Sir Ernest Clark, K.C.B., C.B.E., Governor of Tasmania.

His Excellency Sir Hubert Murray, K.C.M.G., Lieutenant-Governor of Papua.

The Honourable Sir George Murray, K.C.M.G., Chancellor of the University of Adelaide.

His Excellency Captain the Right Honourable Lord Wakehurst, K.C.M.G., Governor of New South Wales.

The Honourable Richard L. Butler, Premier of South Australia.

President:

Sir Henry Simpson Newland, M.B., B.S., F.R.C.S., F.R.C.S., Edinburgh (Honorary), F.R.A.C.S., F.A.C.S.

Vice-Presidents:

Dr. F. Antill Pockley, Wahroonga, New South Wales (President, Australasian Medical Congress, Ninth Session, 1911, Sydney).

Dr. A. C. Purchas, Panmure, Auckland (President, Australasian Medical Congress, Tenth Session, 1914, Auckland).

Sir Louis Barnett, North Otago, New Zealand [President, Australasian Medical Congress (British Medical Association), Second Session, 1927, Dunedin].

Dr. G. H. Abbott, Sydney [President, Australasian Medical Congress (British Medical Association), Third Session, 1929, Sydney].

Dr. D. H. E. Lines, New Town, Tasmania [President, Australasian Medical Congress (British Medical Association), Fourth Session, 1934, Hobart].

Sir James W. Barrett, Melbourne, Past-President of the British Medical Association 103rd Annual Meeting, 1935, Melbourne.

Dr. W. N. Robertson, Vice-President of the British Medical Association, Brisbane.

Dr. R. H. Fetherston, Vice-President of the British Medical Association, Melbourne.

Dr. H. E. Gibbs, Vice-President of the British Medical Association, Wellington, New Zealand.

Surgeon Captain W. J. Carr, R.A.N., Director of Naval Medical Services of Australia, Navy Office, Melbourne.

Major-General R. M. Downes, C.M.G., V.D., Director-General of Medical Services, Australia, Victoria Barracks, Melbourne.

Dr. J. H. L. Cumpston, C.M.G., Director-General of Health of the Commonwealth of Australia, Canberra.

Colonel F. T. Bowerbank, O.B.E., Director of Medical Services, N.Z.M.C., Wellington.

Dr. M. H. Watt, O.B.E., Director-General of Health of the Dominion of New Zealand, Wellington.

Representatives of Branches: Dr. T. A. Price, Queensland; Dr. A. A. Palmer, New South Wales; Dr. J. Newman Morris, Victoria; Dr. F. W. Carter, Western Australia; Dr. G. E. Clemons, Tasmania; Major T. D. M. Stout, Wellington, New Zealand; Dr. H. Swift, South Australia.

Proposed Programme.

The following is the proposed programme for congress week.

Monday, August 23.

Morning.—Registration of members.

Afternoon.—Proposed afternoon reception or garden party; Masonic luncheon at 1 o'clock.

Evening.—Inaugural meeting at Bonython Hall at 8 o'clock (President's address); presentation of the Gold Medal of the British Medical Association (Australia) to Sir Henry Newland; presentation of the Stawell Memorial Clinical Prize.

Tuesday, August 24.

Morning.—Plenary session. Speakers: Dr. M. J. Holmes, Dr. L. B. Bull, Dr. Cotter Harvey. The subject to be discussed will embrace some aspects of tuberculosis.

Afternoon.—12.30 o'clock: Official photograph of congress. 1 p.m.: Ear, Nose and Throat Section luncheon; Ophthalmological Section luncheon.

Evening.—President's reception in Town Hall.

Wednesday, August 25.

Evening.—Public lecture: address by Professor Wood Jones, of Melbourne; Returned Soldiers' dinner; Medical Women's dinner.

Thursday, August 26.

Luncheon for Fellows and Members of the British College of Obstetricians and Gynæcologists.

Evening.—Congress dinner, at which both men and women may attend, to be held at John Martin's Dining Hall.

Friday, August 27.

It is proposed to hold a golf competition for a cup presented by Dr. T. G. Wilson. Further particulars later.

Evening.—Congress dance at Palais Royal.

Church services have been arranged for Sunday, August 22, at Saint Peter's Cathedral, Saint Francis Xavier's Cathedral, Pirie Street Methodist Church.

The Ladies' Executive Committee has arranged numerous excursions in the country, luncheons, late afternoon parties *et cetera*. It has also arranged a golf competition.

THE Honorary Secretary of the Section of Obstetrics and Gynæcology advises that it is proposed to hold a luncheon for Fellows, Members and holders of diplomas of the British College of Obstetricians and Gynæcologists during the Fifth Session of the Australasian Medical Congress (British Medical Association). This function will take place on Thursday, August 26, 1937, and the Honorary Secretary of the Section, Dr. H. M. Fisher, would be glad if members intending to be present would communicate with him as soon as possible, at 178, North Terrace, Adelaide, South Australia.

Royal College of Surgeons of England.

PRIMARY FELLOWSHIP EXAMINATION.

CANDIDATES are reminded that the last date on which entrance forms for the Primary Fellowship Examination of the Royal College of Surgeons of England to be held at Sydney and Melbourne on November 29, 1937, must be lodged is July 31, 1937.

Completed entrance forms must be accompanied by certificates, signed by the Registrar of the University, that the candidates have matriculated and have passed the third-year examinations in anatomy and physiology. Graduates must forward advice of the date of graduation.

Third-year medical students who desire to enter for the examination must lodge provisional entrance forms on or before July 31, 1937, and, at a later date, supply evidence of having passed the third-year examinations in anatomy and physiology.

All entries must be forwarded to the Secretary, Royal Australasian College of Surgeons, Spring Street, Melbourne, C.1.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the *Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Burgess, Clara, M.B., Ch.B., 1933 (Liverpool), Lachlan Park, New Norfolk.

Jones, Lorna Doreen, M.B., B.S., 1931 (Univ. Melbourne), Strahan, Tasmania.

Books Received.

FLUORINE INTOXICATION: A CLINICAL-HYGIENIC STUDY, with a Review of the Literature and Some Experimental Investigations, by K. Roholm; 1937. Copenhagen: Nyt Nordisk Forlag; London: H. K. Lewis and Company Limited. Imperial 8vo, pp. 375, with illustrations.

THE DIAGNOSIS AND TREATMENT OF DIABETES AND THE USE OF HIGH CARBOHYDRATE DIETS, by W. W. Ingram, M.C., M.D.; Second Edition; 1937. Australia: Angus and Robertson Limited. Crown 8vo, pp. 148. Price: 6s. net.

Diary for the Month.

JULY 20.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 20.—Tasmanian Branch, B.M.A.: Council.
JULY 21.—Western Australian Branch, B.M.A.: Branch.
JULY 22.—New South Wales Branch, B.M.A.: Clinical meeting.
JULY 23.—Queensland Branch, B.M.A.: Council.
JULY 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JULY 28.—Victorian Branch, B.M.A.: Council.
JULY 29.—South Australian Branch, B.M.A.: Branch.
JULY 29.—New South Wales Branch, B.M.A.: Branch.
AUG. 3.—New South Wales Branch, B.M.A.: Organization and Science Committee.
AUG. 3.—Tasmanian Branch, B.M.A.: Council.
AUG. 4.—Victorian Branch, B.M.A.: Branch.
AUG. 4.—Western Australian Branch, B.M.A.: Council.
AUG. 5.—South Australian Branch, B.M.A.: Council.
AUG. 6.—Queensland Branch, B.M.A.: Branch.
AUG. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
AUG. 10.—Tasmanian Branch, B.M.A.: Branch.
AUG. 13.—Queensland Branch, B.M.A.: Council.
AUG. 17.—Tasmanian Branch, B.M.A.: Council.
AUG. 17.—New South Wales Branch, B.M.A.: Ethics Committee.

Medical Appointments.

Dr. A. E. W. Burrell has been appointed Government Medical Officer at Griffith, New South Wales.

* * *

Dr. N. E. Kirkwood has been appointed Medical Officer in the Department of Mental Hospitals, New South Wales.

* * *

Dr. I. A. D. Graham has been appointed Medical Officer in the office of the Director-General of Public Health of New South Wales.

* * *

Dr. C. I. Streich has been appointed Medical Officer of Health to the Armadale-Kelmscott Road Board, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser", pages xiv to xvii.

BRISBANE AND SOUTH COAST HOSPITALS BOARD, QUEENSLAND: Honorary Physician.
BROKEN HILL AND DISTRICT HOSPITAL, BROKEN HILL, NEW SOUTH WALES: Resident Medical Officer.
INSTITUTE OF MEDICAL RESEARCH, ADELAIDE, SOUTH AUSTRALIA: "Neale" Research Pathologist.
LACHLAN PARK HOSPITAL, NEW NORFOLK, TASMANIA: Assistant Medical Officer.
MOOROOPNA BASE HOSPITAL, MOOROOPNA, VICTORIA: Junior Resident Medical Officer.
NAREMBEEN ROAD BOARD, NAREMBEEN, WESTERN AUSTRALIA: Medical Officer.
ROYAL AUSTRALIAN AIR FORCE: Medical Officer.
ROYAL MELBOURNE HOSPITAL, MELBOURNE, VICTORIA: Honorary Officers.
THE METHODIST OVERSEAS MISSION, SYDNEY, NEW SOUTH WALES: Medical Officers.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 178, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

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